VOLUME 45 NUMBER 2 FEBRUARY, 1958

AMERICAN JOURNAL OF OPHTHALMOLOGY

THIRD SERIES FOUNDED BY EDWARD JACKSON

CONTENTS

DEPARTMENTS

For a complete table of contents see page xxix

Publication office: 450 Ahnaip St., Menasha, Wisconsin

Subscription price in United States twelve dollars yearly. In Canada and foreign countries fourteen dollars. Published monthly by the Ophthalmic Publishing Company. Subscription and Advertising Office: 664 North Michigan Avenue, Chicago 11, Illinois. Entered as second class matter at the post office at Menasha, Wisconsin. Printed in U.S.A.

Her Best Friend Can't Tell

Your patients gain confidence in their public life knowing that others won't notice the artificial eye. They avoid feelings of inferiority in their personal life when best friends can't tell the difference.

Let our 105 years' experience help you perfectly match your patients' eyes. Our experienced men visit most areas regularly to fit your patients, custom-made eyes to match, and offer you technical assistance on difficult cases. Call or write our nearest office.

Complete Artificial Eye Service

- **Eyes custom made—glass or plastic**
- **Eyes from stock sent on memorandum same day order received—glass or plastic**
- **Damaged or broken artificial eyes accurately matched**
- **Fitted to all types of motility implants**
- **Implants, X-Ray therapy shields, foreign body locators**
- **Superior Quality—Finest Workmanship**

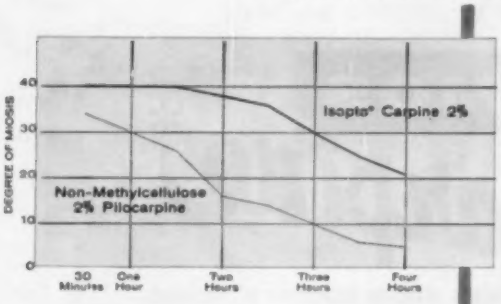
Serving the Profession Since 1851

Mager and Gougelman inc.

30 North Michigan Avenue
Chicago 2, Illinois

120 E. 56th St.
New York 22, New York

DETROIT • CLEVELAND • KANSAS CITY • MINNEAPOLIS • ST. LOUIS
HOUSTON (Soper Bros.) • BOSTON • PHILADELPHIA • PITTSBURGH • WASHINGTON



Prolonged control in glaucoma therapy

Demonstrating the advantages of a methylcellulose menstruum

Laboratory research* demonstrates that ISOPTO® CARPINE effects a greater degree and significantly more prolonged duration of miosis than a non-Methylcellulose solution.

*Animal experimentation

ISOPTO CARPINE Sterile Ophthalmic Solution
PILOCARPINE HYDROCHLORIDE WITH 0.5% METHYLCELLULOSE.
0.25% 0.5% 1% 2% 3% 4%

ISOPTO ESERINE Sterile Ophthalmic Solution
ESERINE SALICYLATE WITH 0.5% METHYLCELLULOSE.
0.25% 0.5%

sterile · well stabilized · minimal irritation · uniform · pH and tonicity adjusted

SUPPLIED: ISOPTO® CARPINE
15cc Drop-Tainer®

ISOPTO® ESERINE
15cc Drop-Tainer®

ISOPTO® DESIGNATES ALCON STERILE OPHTHALMIC PREPARATIONS CONTAINING METHYLCELLULOSE

rhinologic and sterile ophthalmic specialties

Alcon

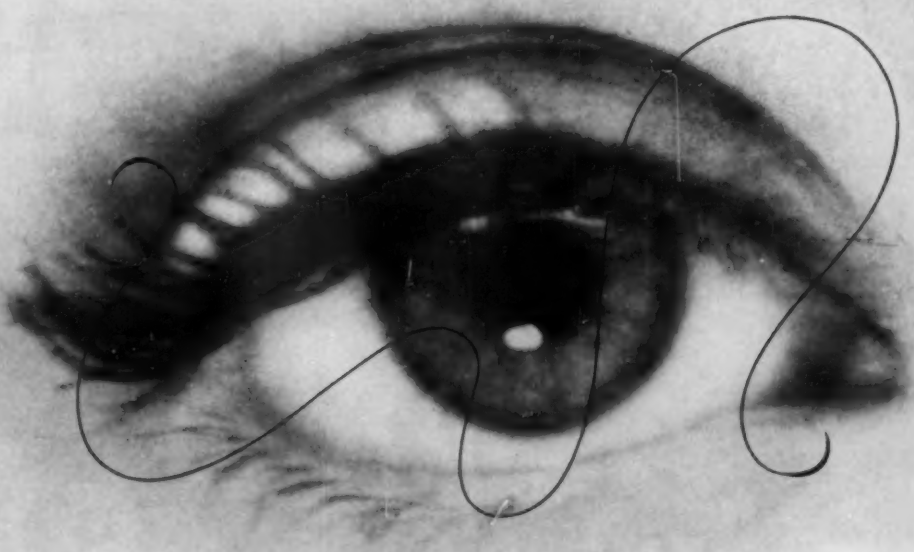
ALCON LABORATORIES, Inc.
FORT WORTH, TEXAS

Alcon products are readily available to pharmacies from their Service Wholesale Drug Suppliers in the U. S. and Canada. Export Dept.: 120 S. LaSalle St., Chicago

CARPINE-1

DESIGNED FOR YOUR MOST EXACTING REQUIREMENTS

ETHICON® OPHTHALMIC NEEDLE SUTURES



- hand-finished Micro-Point® needles
- unequaled sharpness by hand honing
- 40% greater strength
- increased stability in needle holder
- reduced tendency to cut out of tissue

ETHICON EYE SUTURES with Tru-Tempered reverse cutting needles for maximum strength and minimum tendency to cut out of tissue.

ETHICON®

Unsurpassed Comfort in Glaucoma Therapy

PILOMIOTIN

STABILIZED PILOCARPINE HCl.

IN SELF-SEALING
SELF-DISINFECTING
LACRIVIALS

- ✓ *surgical quality*
- ✓ *consistently effective*
- ✓ *non-allergenic*
- ✓ *dated — your assurance of*
  sterility
  stability
  potency

½, 1,
2, 3, 4%

PIONEER SPECIALISTS IN
STERILE OPHTHALMIC SOLUTIONS

ISO-SOL

BROOKLYN 17, N. Y.
MONTREAL — PANAMA

S
sh

And,
of the
liant
perma

In you
Red,
num
. . . a
Engra

Whet
featu
Eyew
DBL
and y

like

She'll look the way she likes-in the new Shurflair Shurset

And, as the picture shows, no wonder. All of the glamour . . . the styling . . . the brilliant gleaming beauty of Ronflair *now with permanized DBL.*

In your choice of Sienna, Ebony, Cranberry Red, Dusk Blue or Golden anodized aluminum colors . . . with or without engraving . . . and with matching Panel, Spinet, Hand Engraved or Jeweled Temples.

Whether you prefer the adjustable DBL feature of *Ronflair*, in either Shurset or Full Eyewire models . . . or the permanized DBL of Shurflair, in Shurset only . . . you and your most fastidious patients will . . .

like the way they look

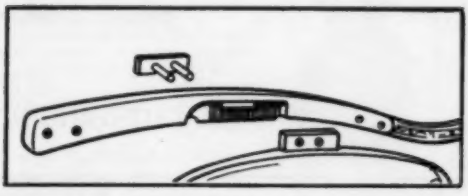
SHURON OPTICAL COMPANY, INC.
Geneva, N. Y., Rochester, N. Y.

SHURON

News About . . .

SHURFLAIR SHURSET

Shurflair's permanized DBL is accomplished, as is shown in the diagram below, by a double rivet which permanently secures the Shurset mounting to the brow-bar. Done at the factory, of course, all that shows is the inconspicuous head of the rivet on the back. Permanizing does not affect the removability of the brow caps for making adjustments or for changing to another color or style of cap.



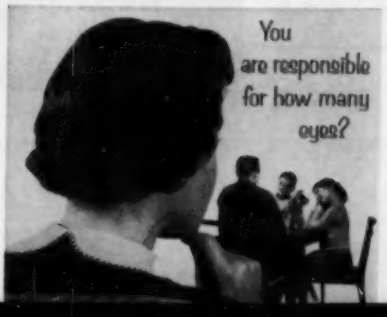
Shurflair is available in Shurset mounting only (the Full Eyewire Unit because of the screw at the top does not lend itself to permanizing), in the following sizes:

42-18, 20, 22, 24
44-18, 20, 22, 24
46-18, 20, 22, 24
48-18, 20, 22, 24

NATIONAL VISUAL CARE ADVERTISING EXTENDED TO LADIES' HOME JOURNAL

In addition to Shuron's continuing campaign in The Saturday Evening Post and Life Magazine, a new series starts in the March Journal—out February 27.

We don't think you will be apt to underestimate the power of this women's campaign . . . you know how important these 20 million wives and mothers are in motivating up-to-date examinations for their children, their husbands—and themselves.



AMERICAN JOURNAL OF OPHTHALMOLOGY
SERIES 3 · VOLUME 45 · NUMBER 2 · FEBRUARY, 1958

CONTENTS

Fig. 1

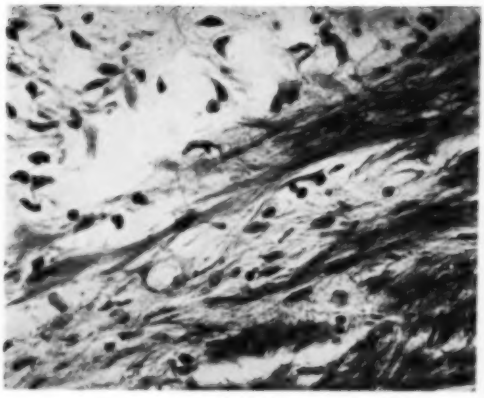


Fig. 2

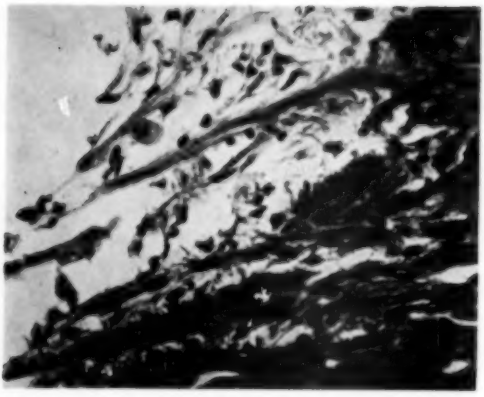


Fig. 3

Fig. 4

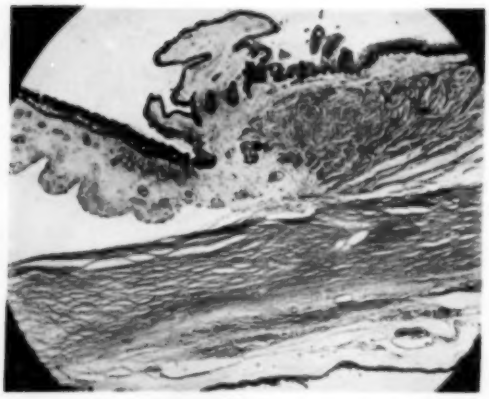


Fig. 5

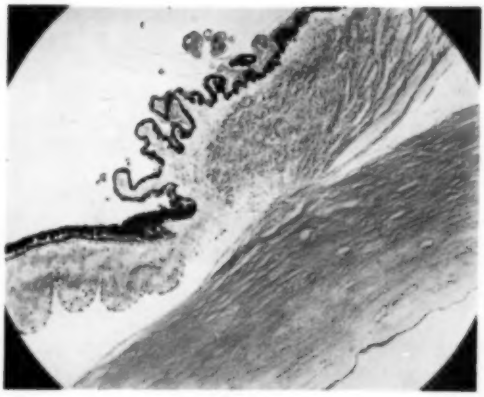


Fig. 6

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 45 FEBRUARY, 1958 NUMBER 2

A MECHANISTIC ELEMENT IN TRABECULAR FUNCTION*

BENJAMIN RONES, M.D.
Washington, D.C.

The past decade has seen an extraordinary advance in the clarification of the mechanisms of glaucoma. A sharp distinction has been drawn between closed-angle glaucoma and chronic simple glaucoma, in which the intraocular pressure is elevated in spite of an open chamber angle. It is easy to understand why an elevation of pressure occurs when the chamber angle becomes occluded from various causes. However, the nature of the obstruction to outflow in chronic simple glaucoma continues to confound even the most ingenious theorists and experimentalists.

Recently, exact studies have been carried out with regard to the trabecular meshwork of the normal human eye. Ashton, Brini, and Smith[1] attempted to determine to what extent structural changes are responsible for an abnormal resistance to aqueous outflow and at which particular sites in the drainage area the impediment is located. In their study the posterior attachments of the trabecular meshwork are mentioned only in a passing reference to the work of Burian, Braley, and Allen.[2]

Flocks[3] at the same time studied the anatomy of the trabecular meshwork as seen in tangential sections. His interest was directed particularly to the size and shape of the openings in the meshwork and the "pore tissue."

Burian, Braley, and Allen[2] observed four groups of trabecular fibers, three of which had attachments to the uveal tract. One group had an inconstant posterior termination; in some instances all of the fibers passed into the tip of the scleral spur, in others a third to half of the fibers merged with the connective tissue surrounding the meridional fibers of the ciliary muscle. Fibers of another group took a more circular course, and terminated posteriorly in the connective tissue surrounding the radial and circular fibers of the ciliary muscle. They also attempted to observe by gonioscopy[4] the mechanical changes in the chamber angle dur-

* From the Laboratory of Ophthalmic Pathology, Episcopal Eye, Ear, and Throat Hospital. Presented at the Wilmer Institute Resident's Association meeting, April 5, 1957.

FIGS. 1 TO 6 (RONES). A MECHANISTIC ELEMENT IN TRABECULAR FUNCTION.

Fig. 1 (A.F.I.P. Neg. No. 57-2272, ×35). The hematoxylin-eosin stained section of this normal angle shows, even at low magnification, that there are insertions of the meridional fibers of the ciliary muscle into the trabecular meshwork.

Fig. 2 (A.F.I.P. Neg. No. 57-2273, ×35). The Masson trichrome stain causes muscle to appear red and collagenous tissue blue. In a section similar to the preceding, the relationship of muscle to meshwork becomes more clearly defined.

Fig. 3 (A.F.I.P. Neg. No. 57-2259, ×195). Higher magnification demonstrates that the muscle fibers attach to the meshwork extensively and directly.

Fig. 4 (A.F.I.P. Neg. No. 57-2279, ×440). The relationship of the muscle fibers to the collagenous bundles resembles that of muscular insertion into a tendon, as is clearly seen in this high magnification of isolated elements.

Fig. 5 (A.F.I.P. Neg. No. 57-2258, ×440). Tangential sections of this area demonstrate the muscle-meshwork relationship to be diffuse and intimate.

Fig. 6 (A.F.I.P. Neg. No. 57-2278, ×320). Another view to demonstrate this broad and intimate relationship.

ing accommodation. They could reach no definite conclusions even though they observed a subtle change in the appearance of the trabecular zone.

These recent studies with modern techniques are all concerned with the diffusion of the aqueous through channels and pores in the trabecular meshwork and Schlemm's canal. No studies have been performed as to propulsive mechanisms due to muscular activity. Numerous conflicting statements are found in the recent literature and these should be assessed.

In the Unesco Symposium, Duke-Elder,[5] in his discussion of the phasic variations of pressure, stated that in the ordinary course of simple glaucoma wherein the angle is wide open the only reasonable hypothesis for the hypotensive action of miotics is facilitation of drainage by a vascular effect. He thought it much less likely that the pull of the ciliary muscles on the trabeculae could have a comparable effect.

Leydhecker[8] on the other hand quoted Fortin and Hruby to the effect that when accommodation is abolished the trabecular spaces and Schlemm's canal become narrow, the arteries of the ciliary body open, and its veins are narrower than when accommodation is active, with the probable result that more aqueous collects in the eye.

Weekers[5,6] expressed the opinion that the action of pilocarpine through traction on the trabeculae in simple glaucoma was only a hypothesis which cannot explain all the facts, nor did he think that the vasomotor changes form a completely satisfactory explanation. He suggested that the pull of the ciliary muscle opens exit channels which are normally closed.

Asayama,[7] in 1902, published a paper with beautiful illustrations of the structure of the trabecular meshwork and its attachments to the ciliary muscle. He stated that his observations tempted him to assume that the contraction of the meridional bundles of the ciliary muscle exerts a certain pull on the pectinate meshwork, thus loosening the meshwork and allowing more aqueous to enter the canal of Schlemm.

Thomson[8] was not able to trace the meridional fibers of the ciliary muscle forward over the tip of the scleral spur, as described by others. His view was that the muscle fibers were attached to the internal and posterior surface of that process, and the elastic trabecular tissue to the anterior surface. Thus while contraction of the meridional fibers will pull the spur backward and inward, the elastic tissue attached to its anterior surface will by its resiliency tend to counteract this and pull it forward and outward again. Such an arrangement would provide the means for lowering and raising the pressure within the canal. When the pressure was negative, fluid from the anterior chamber would be sucked into the canal and then expelled into the venous system when the scleral spur sprang back, thus narrowing the canal and acting as a "pump."

The ciliary muscle is described in standard textbooks (Salzmann's[9] and Duke-Elder's[10]) as being composed of three muscle bundles. Apparently these authors have not accepted the work of Asayama, but agree with Thomson that the meridional fibers have their anterior attachment in the scleral spur. The findings to be reported in this paper will disagree with Thomson.

Asayama's paper was written before photography was being utilized in medical illustration. His drawings are excellent but, since they are drawings, they can be disputed as merely the interpretation of the observing artist and do not represent the true state of affairs. The purpose of this presentation is to demonstrate photographically by the conventional hematoxylin and eosin staining method, and by the Masson stain, that there are definite attachments between the meridional and radial fibers of the ciliary muscle and the trabecular meshwork. This does not presuppose an indirect attachment through the medium of surrounding connective tissue, as claimed by Burian, Braley, and Allen.[2] The accompanying photographs suffice in

establishing this point and need no further description.

Davson[11] has stated that the production of the ocular fluid should be attributed to the ciliary processes on morphologic grounds, but that definite proof of this function is exceedingly difficult to establish, either by histologic or physiologic experiments. He has also called attention to the lack of factual knowledge of the physical factors which determine the outflow of aqueous humor. The fluid-pressure is probably greater than the pressure in that part of the venous system into which the fluid drains, determining the rate of drainage. The question as to whether a large part of the resistance to outflow from the eye is encountered during flow into Schlemm's canal (Goldmann;[12] Bárány[13]) or among the collector and aqueous veins (Perkins;[14] Grant[15]) is as yet unsettled. Nevertheless, these two factors, the pressure-head and the resistance to outflow, determine the rate of outflow of the fluid from the eye.

Comment

These sections demonstrate that the attachment of the meridional and radial muscle fibers to the ciliary meshwork is a real one, and not an anatomic oddity. The muscle fibers blend directly with the trabecular fibers, so that the contraction and relaxation of the former must necessarily cause a change in the tension of the latter.

I propose to theorize from these anatomic facts, that the constant innervational change of muscle tonus must induce a fluctuation of hydrostatic pressure relationships in the spaces of the trabecular meshwork. The continuous process of contraction and relaxation can be visualized as a spongelike action of the meshwork sucking the aqueous into its pores and then squeezing it along its course into Schlemm's canal. The beautiful demonstration by Zimmerman[16] that hyaluronic acid occurs in the trabecular meshwork allows him to speculate whether this substance influences the facility of aqueous outflow, or whether its shock-absorbing and lubricating functions might be important in keeping open the pathways from the anterior chamber to the canal of Schlemm. The marriage of these two views would allow the legitimate conception of a mechanical and chemical sponge facilitating the transmission of the intraocular fluid into the outflow channels. Flocks and Zweng,[17] in a preliminary report, bolster this concept by demonstrating that in monkeys in which the ciliary body was contracted by pilocarpine administration, the openings in the trabecular meshwork were larger and rounder than in an atropinized eye.

Summary

In 1902, Asayama first offered evidence that the attachments of the ciliary body to the trabecular meshwork might have some effect on the flow of the intraocular fluid. This was largely forgotten and other theories of muscular attachment have been dominant. The recent awakening of interest in the chamber angle, and the inability to explain many normal and pathologic behavior characteristics at this site by the newer techniques of chemistry, tonography, and gonioscopy, have led me to paraphrase the bride's jingle:

Something old—Asayama's observations on ciliary muscle attachment.

Something new—The concept of the meshwork as a chemical and mechanical sponge.

Something borrowed—Gonioscopic and tonographic evidence of ciliary muscle activity.

Something blue—Blue-staining hyaluronic acid in the trabecular meshwork.

1302 18th Street, N.W. (6).

References

1. Ashton, N., Brini, A., and Smith, R.: Anatomical studies of the trabecular meshwork of the normal human eye. Brit. J. Ophth., **40**:257-282, 1956.

2. Burian, H. M., Braley, A. E., and Allen, L.: External and gonioscopic visibility of the ring of Schwalbe and the trabecular zone. Tr. Am. Ophth. Soc., **52**:389-428, 1954.
3. Flocks, M.: The anatomy of the trabecular meshwork as seen in tangential section. Arch. Ophth., **56**:708-718, 1956.
4. Burian, H. M., and Allen, L.: Mechanical changes during accommodation observed by gonioscopy. Arch. Ophth., **54**:66-72, 1955.
5. Duke-Elder, S. (Ed.): Glaucoma: A symposium organized by the Council for International organizations of Medical Sciences. Springfield, Ill., Thomas, 1955.
6. Weekers, R., Watillon, M., and de Rudder, M.: Experimental and clinical investigations into the resistance of outflow of aqueous humor in normal subjects. Brit. J. Ophth., **40**:225-233, 1956.
7. Asayama, J.: Zur Anatomie des Ligamentum pectinatum. Arch. f. Ophth., **53**:113-128, 1901-1902.
8. Thomson, A.: The filtration angle. Ophthalmoscope, **9**:470-481, 1911.
9. Salzmann, M.: The Anatomy and Histology of the Human Eyeball. Chicago, Univ. Chicago Press, 1912.
10. Duke-Elder, S.: Textbook of Ophthalmology. London, Kimpton, v. 1, 1932.
11. Davson, H.: Physiology of the Ocular and Cerebrospinal Fluids. Boston, Little, Brown, 1956.
12. Goldmann, H.: Pressure in the canal of Schlemm. Brit. J. Ophth., **39**:764, 1955.
13. Bárány, E. H.: In vitro studies of the resistance to flow through the angle of the anterior chamber. Acta Soc. Med. Upsaliensis, **59**:260-276, 1953.
14. Perkins, E. S.: Pressure in the canal of Schlemm. Brit. J. Ophth., **39**:215-219, 1955.
15. Grant, W. M.: Facility of flow through the trabecular meshwork. Arch. Ophth., **54**:245-248, 1955.
16. Zimmerman, L. E.: Demonstration of hyaluronidase-sensitive acid mucopolysaccharide: In trabecula and iris in routine paraffin Sections of adult human eyes. Am. J. Ophth., **44**:1 (July) 1957.
17. Flocks, M., and Zweng, H. C.: The effect of cyclotonia and cycloplegia on the histology of the trabecular meshwork of monkey eyes. Am. J. Ophth., **43**:294, 1957.

ON CHANGES IN INTRAOCULAR PRESSURE AFTER APPLICATION OF THE TONOMETER*

In the same eye and in the other eye

Frederick W. Stocker, M.D.
Durham, North Carolina

Ever since the observation of Pagenstecher,[1] in 1878, that massage of the eye would reduce the intraocular pressure, this fact has periodically aroused the interest of ophthalmologists. Polak-van Gelder,[2] in 1911, reported that the drop of pressure after massage was more marked in normal than in glaucomatous eyes, and Paul Knapp,[3] in 1912, stated that the pressure-reducing effect was of longer duration in normal than in glaucomatous eyes. After Böck, Kronfeld, and Stough[4] had studied this phenomenon again in 1934, Grant[5] and Moses and Bruno[6] developed, based on those previously known facts, the method of measuring the aqueous outflow now known as tonography.

In tonography it is assumed that the difference between the intraocular pressure found in the beginning and at the end of the four-minute period, during which the tonometer rests on the eye, is indicative of the amount of aqueous forced out of the chamber angle. According to Posner[7] the weight of the tonometer is 16.5 gm. The total weight applied during tonometry therefore varies from 22 to 26.5 gm. according to the various weights that are added to the plunger.

While there is ample evidence that by applying this weight on the cornea fluid is forced out through the chamber angle at an increased rate, the possibility of additional effects on the eye have to be considered. This possibility, although not completely ignored, has not been adequately investigated by most authors working on tonography.

Amsler and co-workers,[8] however, raised

---

* From the McPherson Hospital and Duke University School of Medicine. Presented at the 92nd annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, June, 1956.

the question in their recent monograph on aqueous humor, "What happens in the eye, a living organ, besides the indisputable and not disputed expression of aqueous humor under the continuous weight of the tonometer—?"

Indeed, it is hardly conceivable that no other reaction would occur in this extremely delicately organized and sensitive organ when it is subject to such an unphysiologic pressure. Furthermore, while in certain types of glaucoma tonographic findings are consistent with the theory of increased resistance to aqueous outflow, there are cases in which the readings are not compatible with the usual clinical differentiation between normal and glaucomatous eyes. The occurrence of so-called false positives, on which I have previously commented,[9] are one example. The limits of clinical application of tonography have indeed been brought to light ably by Scheie and co-workers.[10]

In order to obtain some evidence of changes, other than fluid being forced out through the chamber angle, that might occur in both eyes when one eye is subject to tonography we determined the difference in intraocular pressure if:

1. The second reading follows the first one immediately (approximately 30 seconds).
2. The second reading follows the first one after four minutes.
3. The second reading follows the first one after four minutes while the tonometer has been resting on the other eye.

The standard Mueller electronic tonometer was used for all measurements. The results are recorded in Table 1.

It appears that the difference in intraocular pressure between the first and second reading is null or minimal if the second reading follows the first one within 30 seconds. The average reduction found at the second reading is 0.97 mm. Hg. In 12 of 20 cases no difference in the two readings was found.

If tonometry is repeated four minutes after the first reading, the average reduction in intraocular pressure is much greater, 1.9 mm. Hg.*

TABLE 1

CHANGES IN INTRAOCULAR PRESSURE AFTER TONOMETRY AND TONOGRAPHY

(Total: 60 cases)

If Tonometry is Repeated	Average Reduction in Intraocular Pressure (in mm. Hg)
1. On the same eye within 30 seconds after the first reading (20 cases)	0.97
2. On the same eye four minutes after the first reading (20 cases)	1.90
3. On one eye after the tonometer has been resting on the other eye for four minutes (20 cases)	2.72
a. On nonglaucomatous eyes (12)	2.37
b. On glaucomatous eyes (8)	3.25

The data contained in this table were statistically analysed and found to be highly significant.

If tonometry is repeated in the left eye, for instance, four minutes after the first reading, while the right eye was subject to the weight of the tonometer during that four-minute period, the difference is even greater. The average reduction for the second reading is 2.72 mm. Hg for the whole group. For the group of nonglaucomatous eyes[12] it is 2.37 mm. Hg; for the group of glaucomatous eyes[8] it is 3.25 mm. Hg. In order to determine whether the difference between the nonglaucomatous and the glaucomatous eyes is significant, a larger series of cases will have to be studied.

DISCUSSION

The first question I pose is:

Can the difference between the results in the first group (second reading within 30

* While this paper was in print, a series of 10 cases was done with the patient sitting up immediately after the first tonometry. The difference in intraocular pressure after four minutes was even greater; that is, 2.57 mm. Hg on the average. This should exclude the possibility of the influence of circulatory changes due to the recumbent position.

seconds) and the second group (second reading after four minutes) be explained by the simple fact that fluid is forced out through the chamber angle by the pressure of the tonometer?

The answer has to be in the negative.

It is perfectly obvious that fluid is expressed by the pressure of the tonometer while the latter is resting on the cornea. There is no reason, however, for continued increase in outflow after the pressure is removed. On the contrary, one would rather expect that the intraocular pressure would increase during that period as the normal inflow of aqueous should gradually replace the aqueous lost by the first application of the tonometer. My observation is in keeping with the well-known fact that a blunt trauma to the eye often results in marked hypotony. Certainly forcing aqueous out by the initial blow would not explain the prolonged persistence of hypotony in such cases.

While other investigators have mentioned the fact that the pressure may fall in the other eye while one eye is subject to tonography, this surprising fact has never been studied systematically in humans. As my study was underway, Kornblueth and Linner[11] reported observations made with rabbits that are consistent with my findings.

All these observations make it evident that the application of the tonometer weighing 22 gm. or more on the eye induces changes in the intraocular pressure in the same eye, as well as in the other eye. This phenomenon cannot be explained by the simple mechanical factor of forcing fluid out through the chamber angle alone. While it is difficult to establish just what happens in the eye under such conditions, the most logical explanation would be that, through the mechanism of reflexes, the amount of aqueous formation might be changed. I am in agreement with Kronfeld[12] when he states that "the failure of the fluorometric method to demonstrate phasic variations in aqueous secretions should not be interpreted as evidence against the occurrence of such variations."

The mechanism of the passing of fluorescein from the blood into the aqueous is not necessarily a definite indicator of the rate of aqueous secretion. The long-known diurnal variations of the intraocular pressure have been found by Stepanik[13] to be present also in tonographic readings. This fact also would be explained most satisfactorily by changes in aqueous production since the mechanical factors controlling the resistance to outflow are more likely to be constant. Nevertheless, the possibility of variations in outflow resistance in some cases also has to be considered.

My observations suggest the existence of a local mechanism for regulating the intraocular pressure which might work in the following way: The formation of aqueous is adjusted to a given outflow resistance, maintaining a physiologic intraocular pressure. If the intraocular pressure rises, either due to increased outflow resistance or, as in tonography, by applying a weight on the eye, the production of aqueous is reduced. Glaucoma, that is, abnormally high intraocular pressure with harmful effects on the eye, results when either the resistance to outflow is increased to a point that reduction of aqueous formation within the scope of the regulating mechanism is not sufficient to keep the pressure within normal limits or when the regulating mechanism does not function properly.

The existence of a reflex mechanism acting from one eye to the other has also been demonstrated. Such a mechanism has long been suspected from various clinical experiences, the most dramatic one being the development of an acute attack of glaucoma in the other eye while an operation for glaucoma is performed on one eye.

More difficult to explain are our observations in a few individual cases which did not fall into the general pattern of the group just reported. In contrast to the observations in this group, we found a rise in pres-

sure in one normal and one glaucomatous eye after the other eye had been subject to the tonometer weight for four minutes. An unchanged pressure was found in one eye and a rise of 3.0 mm. Hg in another eye after the tonometer had rested on it for four minutes, neither one showing any clinical signs of glaucoma.

It is conceivable that the deformation which the globe undergoes during tonography might in itself lead to an obstruction of the outflow channels under certain anatomic conditions. This would offer an explanation for the last mentioned cases (false positives). Perhaps sections of eyes that were frozen while subject to the tonometer weight might help to answer this question.

Conclusions

The observations made in this study demonstrate that the mechanism of intraocular pressure is affected in other ways besides the forcing out of fluid through the chamber angle when the eye is subject to the weight of the tonometer. This statement is not made with the intention to minimize the value of the splendid work which has been done in the field of tonography. Important information has been obtained with this method. It may be well, however, to be aware of the extremely intricate physiology of the eye and not to overestimate the role of mechanics alone when using tonography for clinical evaluation.

Summary

Three series of tonometric studies were done with the following results:

1. If tonometry is immediately repeated, the intraocular pressure is found to be identical or only slightly reduced on the second reading.

2. If tonometry is repeated in four minutes, the intraocular pressure is found to be definitely lower.

3. If the second reading is taken four minutes after the first one, while the tonometer has been resting on the other eye during the whole period (tonography), the pressure is found to be even lower.

The meaning of these findings is discussed, and it is concluded that applying a weight of 22 or more gm. to an eye for four minutes must have effects in addition to forcing fluid out through the chamber angle. Some of the clinical observations incompatible with the current theory of tonography may be explained by these side effects.

1110 West Main Street.

I am indebted to Dr. George S. Eadie, professor of physiology and pharmacology in the School of Medicine at Duke University, for the statistical analysis of my data. The averages reported were found to be statistically highly significant with 19 freedoms for the groups of 20.

References

1. Pagenstecher, H.: Ueber die Massage des Auges u. deren Anwendung bei verschiedenen Angenerkrankungen. Centralbl. f. prakt. Augenh., **2**:281-289, 1978.
2. Polak-van Gelder, R. E. S.: Untersuchungen mit dem Tonometer von Schioetz. Klin. Monatsbl. f. Augenh., **49**:592-605, 1911.
3. Knapp, P.: Ueber den Einfluss der Massage auf die Tension normaler and glaucomatoser Augen. Klin. Monatsbl. f. Augenh., **50**:691-716, 1912.
4. Böck, J., Kronfeld, P. C., and Stough, J. T.: Effect on intraocular tension of corneal massage with tonometer of Schioetz. Arch. Ophth., **11**:797-806, 1934.
5. Grant, W. M.: Tonographic method for measuring the faculty and rate of aqueous flow in human eyes. Arch. Ophth., **44**:204-214, 1950.
6. Moses, R. A., and Bruno, M.: The rate of outflow of fluid from the eye under increased pressure. Am. J. Ophth., **33**:388-397, 1950.
7. Posner, A.: The "tone-weight": An ocular compressor. Tr. Am. Acad. Ophth., **59**:544, 1955, and personal communication.
8. Amsler, M., Verry, F., and Huber, A.: L'humeur aqueuse et ses Fonctions. Paris, Masson, 1955.
9. Stocker, F. W.: Discussion of paper by Scheie, Spencer and Helmick.[20]

10. Scheie, H. G., Spencer, R. W., and Helmick, E. D.: Tonography in the clinical management of glaucoma. Tr. Am. Ophth. Soc., **53**:267-299, 1955.
11. Kornblueth, W., and Linner, E.: Experimental tonography in rabbits. Arch. Ophth., **54**:717-724, 1955.
12. Kronfeld, P. C.: A tonographic study of glaucoma. Am. J. Ophth., **39**:147-152, 1955.
13. Stepanik, T.: Diurnal tonographic variations and their relation to visible aqueous outflow. Am. J. Ophth., **38**:629-645, 1954.

CLINICAL TONOGRAPHY: THE SCLERAL RIGIDITY CORRECTION*

ROBERT A. MOSES, M.D., AND BERNARD BECKER, M.D.
Saint Louis, Missouri

Tonography has provided a valuable clinical tool for the diagnosis and evaluation of glaucoma.[1,2] One of the major sources of error in routine tonometry stems from the failure to correct results in the individual eye for its scleral rigidity.[2,3] This error is greatly accentuated in tonography.[3,4] It is the purpose of the present report (1) to present new simplified tables for the rapid estimation of intraocular pressure and outflow facility for eyes of average scleral rigidity based upon the 1955 Friedenwald tables[5] and (2) to offer two methods of applying a correction for deviations from average values for scleral rigidity.

* From the Department of Ophthalmology, Washington University School of Medicine, and the Oscar Johnson Institute. This investigation was supported in part by a research grant, B-621, from the National Institute of Neurological Diseases and Blindness of the National Institutes of Health, Public Health Service.

The research relating to this study was also financed in part under a grant to Washington University School of Medicine made by the Alfred P. Sloan Foundation, Inc. The grant was made upon recommendation of the Council for Research in Glaucoma and Allied Diseases. Neither the foundation nor the council assumes any responsibility for the published findings of this study.

Since this article was accepted for publication, a similar graphic analysis has appeared in an article by E. G. A. van Beuningen and F. W. Fischer (Klin. Monatsbl. f. Augenh., **131**:57-61, 1957; abstract: Am. J. Ophth., **44**:706 (Nov. Pt. I) 1957). However, these authors use the graphic method in their computation of pulse volume of the eye rather than in correction for scleral rigidity in tonography.

A. SIMPLIFIED TONOGRAPHIC TABLES FOR EYES OF AVERAGE SCLERAL RIGIDITY (Tables 1, 2, and 3)

Charts are presented for the 5.5, 7.5, and 10-gm. weights based upon the 1955 tables of Friedenwald. They have been in use in this laboratory for the past three years and have proved convenient for technicians and residents, as well as ophthalmologists. Values for intraocular pressure (P_o) for various scale readings (R) are presented in the lefthand column. The coefficient of outflow facility is readily determined on the basis of the initial reading (R_1) and the number of scale units ($^{\Delta}R$) the tonometer has changed in four minutes. Corrections for the average increase in episcleral venous pressure incident to tonometry (1.25 mm. Hg) have been incorporated in the data.[6]

Thus, if the tonogram with the 5.5-gm. weight begins at a scale reading of 5.0 and reaches a scale reading of seven in four minutes, ($^{\Delta}R = 2.0$) the intraocular pressure is read as 17 mm. Hg and facility of outflow as 0.17 from Table 1. Quarter scale units can be readily estimated by interpolation. For example, if the tonogram started at 5.0 and reached 7.25 at four minutes, the change in scale reading of 2.25 units would be interpreted as an outflow of 0.20.

Empirically, it has proved necessary to use sufficient load on the tonometer so that the initial scale reading is greater than 3.0.

TABLE 1

SIMPLIFIED TONOGRAPHIC TABLE FOR EYES OF AVERAGE SCLERAL RIGIDITY

Initial Reading		5.5-gm. Weight ΔR (Change in Scale Reading)										
P_0	R (5.5 gm.) weight	0	0.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
38	0.50	0	0.06	0.14	0.24	0.39	0.61	0.94				
36	0.75	0	0.06	0.13	0.22	0.35	0.53	0.78				
35	1.00	0	0.05	0.12	0.21	0.32	0.47	0.66	0.94			
33	1.25	0	0.05	0.12	0.19	0.29	0.42	0.59	0.81			
32	1.50	0	0.05	0.11	0.18	0.27	0.38	0.53	0.71	0.94		
30	1.75	0	0.05	0.11	0.17	0.25	0.35	0.48	0.64	0.83		
29	2.00	0	0.05	0.10	0.16	0.24	0.33	0.44	0.58	0.74	0.93	
28	2.25	0	0.05	0.10	0.16	0.23	0.31	0.41	0.53	0.68	0.84	
27	2.50	0	0.04	0.09	0.15	0.22	0.30	0.39	0.49	0.62	0.76	0.92
25	2.75	0	0.04	0.09	0.15	0.21	0.28	0.37	0.46	0.58	0.70	0.84
24	3.00	0	0.04	0.09	0.14	0.20	0.27	0.35	0.44	0.54	0.65	0.78
23	3.25	0	0.04	0.09	0.14	0.20	0.26	0.33	0.42	0.51	0.62	0.74
22	3.50	0	0.04	0.08	0.13	0.19	0.25	0.32	0.40	0.49	0.59	0.70
22	3.75	0	0.04	0.08	0.13	0.19	0.25	0.31	0.38	0.47	0.56	0.66
21	4.00	0	0.04	0.08	0.13	0.18	0.24	0.30	0.37	0.45	0.54	0.63
20	4.25	0	0.04	0.08	0.13	0.18	0.24	0.30	0.36	0.43	0.52	0.60
19	4.50	0	0.04	0.08	0.12	0.17	0.23	0.29	0.35	0.42	0.50	0.58
18	4.75	0	0.04	0.08	0.12	0.17	0.23	0.28	0.34	0.41	0.48	0.56
17	5.00	0	0.04	0.08	0.12	0.17	0.22	0.27	0.33	0.40	0.47	0.54
17	5.25	0	0.04	0.08	0.12	0.17	0.22	0.27	0.33	0.39	0.46	0.53
16	5.50	0	0.04	0.08	0.12	0.16	0.21	0.26	0.32	0.38	0.45	0.52
15	5.75	0	0.04	0.08	0.12	0.16	0.21	0.26	0.32	0.38	0.44	0.50
15	6.00	0	0.03	0.07	0.11	0.15	0.20	0.25	0.31	0.37	0.43	0.49
14	6.25	0	0.03	0.07	0.11	0.15	0.20	0.25	0.31	0.37	0.43	0.49
13	6.50	0	0.03	0.07	0.11	0.15	0.20	0.25	0.30	0.36	0.42	0.48
13	6.75	0	0.03	0.07	0.11	0.15	0.20	0.24	0.30	0.36	0.41	0.47
12	7.00	0	0.03	0.07	0.11	0.15	0.20	0.24	0.29	0.35	0.40	0.46
11	7.50	0	0.03	0.07	0.11	0.15	0.19	0.24	0.29	0.34	0.39	0.45
10	8.00	0	0.03	0.07	0.11	0.15	0.19	0.24	0.29	0.34	0.39	0.45
9	8.50	0	0.03	0.07	0.11	0.15	0.19	0.23	0.28	0.33	0.39	
9	9.00	0	0.03	0.07	0.11	0.15	0.19	0.23	0.28	0.33		
8	9.50	0	0.03	0.07	0.11	0.15	0.19	0.23	0.28			
7	10.00	0	0.03	0.07	0.11	0.15	0.19	0.23				
6	11.00	0	0.03	0.07	0.11	0.15						

At scale readings less than 3.0, falsely elevated pressure and facility values are obtained. Recently, these empirical observations have been subjected to investigation and have proved to depend upon mechanical factors related to tonometer construction.[7]

B. CORRECTION FOR SCLERAL RIGIDITY

It is not the purpose of this paper to advocate routine corrections for variations from average scleral rigidity in tonography because accurate estimation of scleral rigidity is difficult to accomplish. However, with improvement in techniques and

TABLE 2

SIMPLIFIED TONOGRAPHIC TABLE FOR EYES OF AVERAGE SCLERAL RIGIDITY

Initial Reading		7.5-gm. Weight ΔR (Change in Scale Reading)										
P_0	R (7.5 wt.)	0	0.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
54	0.50	0	0.05	0.12	0.23	0.41	0.76					
52	0.75	0	0.05	0.12	0.21	0.36	0.63					
50	1.00	0	0.04	0.11	0.19	0.32	0.50	0.79				
48	1.25	0	0.04	0.10	0.17	0.29	0.44	0.65				
46	1.50	0	0.04	0.09	0.16	0.26	0.38	0.55	0.79			
44	1.75	0	0.04	0.09	0.15	0.24	0.34	0.48	0.67			
42	2.00	0	0.04	0.09	0.14	0.22	0.31	0.43	0.58	0.78		
41	2.25	0	0.04	0.09	0.14	0.20	0.29	0.39	0.52	0.68		
39	2.50	0	0.04	0.08	0.13	0.19	0.27	0.36	0.47	0.60	0.77	
37	2.75	0	0.04	0.08	0.13	0.18	0.25	0.33	0.43	0.54	0.68	
36	3.00	0	0.03	0.07	0.12	0.17	0.23	0.31	0.40	0.50	0.62	0.76
34	3.25	0	0.03	0.07	0.12	0.17	0.22	0.29	0.37	0.46	0.57	0.69
33	3.50	0	0.03	0.07	0.11	0.16	0.21	0.28	0.35	0.43	0.53	0.63
32	3.75	0	0.03	0.07	0.11	0.16	0.21	0.26	0.33	0.41	0.49	0.59
30	4.00	0	0.03	0.06	0.10	0.15	0.20	0.25	0.32	0.39	0.46	0.55
29	4.25	0	0.03	0.06	0.10	0.15	0.19	0.25	0.30	0.37	0.44	0.52
28	4.50	0	0.03	0.06	0.10	0.14	0.18	0.24	0.29	0.35	0.42	0.50
27	4.75	0	0.03	0.06	0.10	0.14	0.18	0.23	0.28	0.34	0.40	0.47
26	5.00	0	0.03	0.06	0.10	0.13	0.17	0.22	0.27	0.33	0.39	0.45
25	5.25	0	0.03	0.06	0.10	0.13	0.17	0.22	0.27	0.32	0.38	0.43
24	5.50	0	0.03	0.06	0.09	0.13	0.16	0.21	0.26	0.31	0.37	0.42
23	5.75	0	0.03	0.06	0.09	0.13	0.16	0.21	0.26	0.31	0.36	0.41
22	6.00	0	0.03	0.06	0.09	0.12	0.16	0.20	0.25	0.30	0.35	0.40
21	6.25	0	0.03	0.06	0.09	0.12	0.16	0.20	0.25	0.29	0.34	0.39
20	6.50	0	0.03	0.05	0.09	0.12	0.15	0.19	0.24	0.28	0.33	0.38
19	6.75	0	0.03	0.05	0.09	0.12	0.15	0.19	0.24	0.28	0.33	0.38
18	7.00	0	0.03	0.05	0.08	0.12	0.15	0.19	0.23	0.27	0.32	0.37
17	7.50	0	0.03	0.05	0.08	0.12	0.15	0.19	0.23	0.27	0.31	0.36
16	8.00	0	0.03	0.05	0.08	0.11	0.15	0.18	0.22	0.26	0.30	0.35
14	8.50	0	0.03	0.05	0.08	0.11	0.15	0.18	0.22	0.26	0.30	
13	9.00	0	0.03	0.05	0.08	0.11	0.15	0.18	0.22	0.25		
12	9.50	0	0.03	0.05	0.08	0.11	0.15	0.18	0.22			
11	10.00	0	0.03	0.05	0.08	0.11	0.14	0.18				
10	11.00	0	0.03	0.05	0.08	0.11	0.14					

the use of applanation tonometry, more accurate estimates are becoming available. Therefore, it seems appropriate to outline methods for applying suitable corrections to selected cases.

The coefficient of scleral rigidity (E) may be measured by the use of the Schiøtz tonometer with two plunger loads, or preferably by the use of the Goldmann applanation tonometer followed by the Schiøtz tonometer with one weight. In tonography this is perhaps best accomplished by doing applanation measurements first, followed by the tonogram with

TABLE 3

SIMPLIFIED TONOGRAPHIC TABLE FOR EYES OF AVERAGE SCLERAL RIGIDITY

Initial Reading		10-gm. Weight ΔR (Change in Scale Reading)										
P_0	R (10 wt.)	0	0.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
75	0.50	0	0.05	0.12	0.26	0.56						
72	0.75	0	0.05	0.11	0.22	0.44						
69	1.00	0	0.04	0.10	0.19	0.35	0.64					
66	1.25	0	0.04	0.10	0.17	0.30	0.50					
64	1.50	0	0.04	0.09	0.15	0.26	0.41	0.66				
61	1.75	0	0.04	0.09	0.14	0.23	0.35	0.54				
59	2.00	0	0.03	0.08	0.13	0.20	0.31	0.45	0.66			
57	2.25	0	0.03	0.08	0.12	0.18	0.28	0.39	0.55			
55	2.50	0	0.03	0.07	0.11	0.17	0.25	0.35	0.47			
53	2.75	0	0.03	0.07	0.11	0.16	0.23	0.32	0.42			
51	3.00	0	0.03	0.06	0.10	0.15	0.21	0.29	0.38	0.49		
49	3.25	0	0.03	0.06	0.10	0.14	0.20	0.27	0.35	0.44		
47	3.50	0	0.03	0.06	0.09	0.13	0.19	0.25	0.32	0.40	0.50	
45	3.75	0	0.03	0.06	0.09	0.13	0.18	0.23	0.30	0.37	0.46	
43	4.00	0	0.02	0.05	0.08	0.12	0.17	0.22	0.28	0.35	0.43	0.52
42	4.25	0	0.02	0.05	0.08	0.12	0.17	0.21	0.26	0.33	0.40	0.48
40	4.50	0	0.02	0.05	0.07	0.11	0.16	0.20	0.25	0.31	0.38	0.44
38	4.75	0	0.02	0.05	0.07	0.11	0.16	0.20	0.24	0.29	0.36	0.42
37	5.00	0	0.02	0.05	0.07	0.10	0.15	0.19	0.23	0.28	0.34	0.40
36	5.25	0	0.02	0.05	0.07	0.10	0.15	0.19	0.23	0.27	0.32	0.38
34	5.50	0	0.02	0.05	0.07	0.10	0.14	0.18	0.22	0.26	0.31	0.36
33	5.75	0	0.02	0.05	0.07	0.10	0.14	0.18	0.22	0.25	0.30	0.34
32	6.00	0	0.02	0.04	0.07	0.10	0.13	0.17	0.21	0.24	0.29	0.33
31	6.25	0	0.02	0.04	0.07	0.10	0.13	0.17	0.21	0.24	0.28	0.32
29	6.50	0	0.02	0.04	0.07	0.10	0.13	0.16	0.20	0.23	0.27	0.31
28	6.75	0	0.02	0.04	0.07	0.10	0.13	0.16	0.20	0.23	0.27	0.31
27	7.00	0	0.02	0.04	0.07	0.09	0.12	0.15	0.19	0.22	0.26	0.30
26	7.25	0	0.02	0.04	0.07	0.09	0.12	0.15	0.19	0.22	0.26	0.30
25	7.50	0	0.02	0.04	0.07	0.09	0.12	0.15	0.18	0.21	0.25	0.29
24	7.75	0	0.02	0.04	0.07	0.09	0.12	0.15	0.18	0.21	0.25	0.29
23	8.00	0	0.02	0.04	0.06	0.09	0.12	0.14	0.18	0.21	0.24	0.28
21	8.50	0	0.02	0.04	0.06	0.09	0.11	0.14	0.18	0.20	0.23	0.27
20	9.00	0	0.02	0.04	0.06	0.09	0.11	0.14	0.17	0.20	0.23	0.26
18	9.50	0	0.02	0.04	0.06	0.09	0.11	0.14	0.17	0.20	0.23	0.26
16	10.00	0	0.02	0.04	0.06	0.09	0.11	0.14	0.17	0.19	0.22	0.26
14	11.00	0	0.02	0.04	0.06	0.09	0.11	0.14	0.17	0.19	0.22	0.25

the appropriate weight. In this fashion prior measurements do not interfere with the subsequent tonogram. The use of the applanation reading and the initial scale reading of the tonogram provides an estimate of scleral rigidity. Caution must be exercised, however, to avoid, or apply suitable correction for, the variation of intraocular pressure with position of the eye with reference to the heart. Thus intra-

ocular pressure may rise one to three or more mm. Hg when lying down if no pillow is used during tonography.[8] Under these circumstances direct comparison of the applanation (sitting) and Schiøtz readings cannot be made.

The application of scleral rigidity estimate to a correction of the outflow facility coefficient may be accomplished by graphic or tabular methods.

1. Graphic method

A graphic representation of measurements for determination of the scleral rigidity coefficient as well as the intraocular pressure and outflow facility coefficient can be made on a Friedenwald nomogram.* In Figure 1, A represents the intraocular pressure as measured with the applanation tonometer (volume displacement 0.6 cu. mm.). R_{10} and $R_{5.5_1}$ represent the scale readings with the 10-gm. and the 5.5-gm. plunger loads, respectively. Any pair of these points defines the oblique line $P_o - R$ the slope of which represents the scleral rigidity for the eye in question. The scleral rigidity line crosses the pressure axis at P_o, the intraocular pressure in the undisturbed eye. Applanation gives a more precise measurement of P_o and per-

* Nomograms may be obtained in pad form from the Ostertag Optical Company, 3851 Washington, Saint Louis, Missouri.

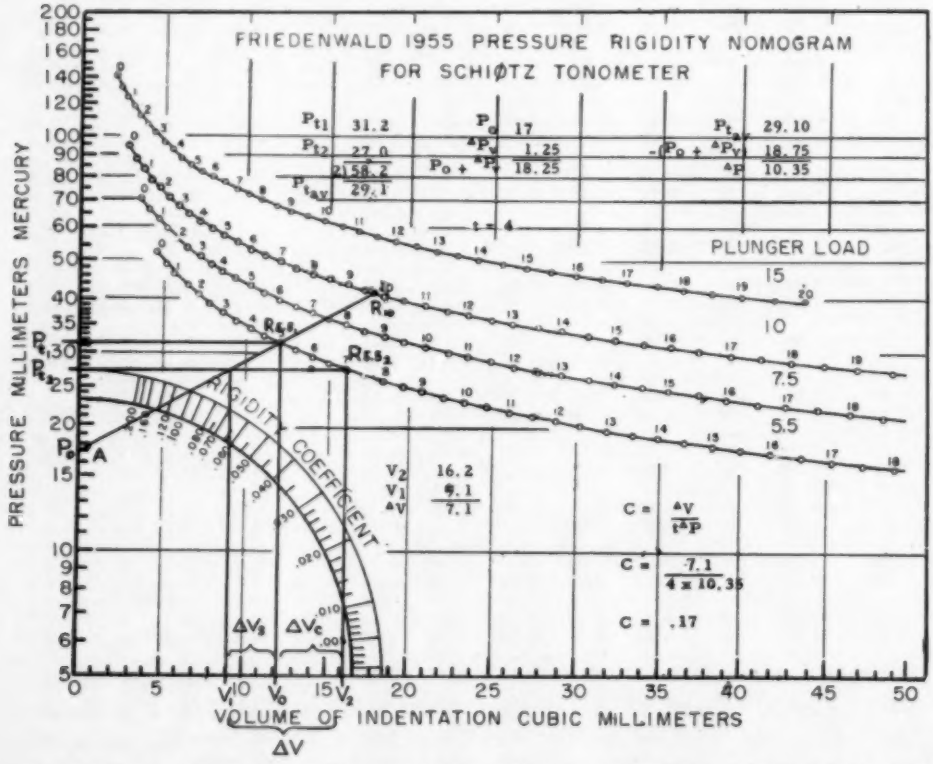


Fig. 1 (Moses and Becker). Graphic representation of tonogram with 5.5-gm. weight where initial scale reading was 5.0 and four-minute reading was 7.0, assuming a normal value for the scleral rigidity coefficient (E = 0.0215).

mits better definition of the line P_oR.[9] At the beginning of tonography ($R_{5.5_1}$) the intraocular pressure with the tonometer in place is P_{t_1}. At the end of tonography ($R_{5.5_2}$) the intraocular pressure with the tonometer in place has fallen to P_{t_2}.

At the beginning of tonography the tonometer has indented the cornea and displaced fluid of volume V_o, but no fluid has been lost from the eye. During tonography fluid is expelled from the eye with a corresponding pressure drop from P_{t_1} to P_{t_2}. This pressure drop corresponds to a volume loss of $V_o - V_1$ or $^{\Delta}V_2$. Also during this pressure drop the corneal indentation has deepened from V_o to V_2 ($^{\Delta}V_c$). The total volume loss during tonography then is $V_2 - V_1 = {}^{\Delta}V$.

The expression used in actually arriving at a value of C is

$$C = \frac{{}^{\Delta}V}{t(P_{t_{av}} - P_o - {}^{\Delta}P_v)}$$

In terms of the graph

$${}^{\Delta}V = V_2 - V_1 \text{ and } P_{t_{av}} = \frac{P_{t_1} + P_{t_2}}{2}.$$

P_o is read directly from the graph. $^{\Delta}P_v$ is taken as 1.25 mm. Hg. t is the duration of the tonogram in minutes.

Examples of the application of this graphic method to the tonogram where the initial scale reading was 5.0 and the four-minute reading was 7.0 are provided in Figures 1 (E = 0.0215), 2 (E = 0.0137), and 3 (E = 0.0342).

2. Tabular method

$$C = \frac{0.0215{}^{\Delta}V_G + (E - 0.0215){}^{\Delta}V_c}{4E(P_{t_{av}} - P_{o_{corr}} - 1.25)}$$

in which

- C is the coefficient of facility of outflow (mm./min./mm. Hg)
- $^{\Delta}V_G$ is the change in ocular volume according to Grant (Table 4)
- E is the coefficient of scleral rigidity as determined clinically (Table 5 or nomogram)
- $^{\Delta}V_c$ is the change in corneal indentation volume during tonography (Table 6)
- $P_{t_{av}}$ is the average of initial and final intraocular pressure during tonography (Table 7)

TABLE 4[5]

Change in ocular volume ($^{\Delta}V$ in cu. mm.) for various plunger weights when the tonometer reading falls from 0 to R

$$^{\Delta}V = \frac{1}{K} \log \frac{P_{to}}{P_{tR}} + V_{oR} - V_{oo}{}^*$$

Scale Reading R	Plunger load 5.5 gm.	7.5 gm.	10 gm.	15 gm.
0.0	0	0	0	0
0.5	1.84	1.93	1.69	1.58
1.0	3.66	3.49	3.32	3.11
1.5	5.44	5.18	4.93	4.61
2.0	7.21	6.85	6.53	6.09
2.5	8.95	8.52	8.11	7.54
3.0	10.69	10.17	9.66	8.97
3.5	12.41	11.81	11.22	10.40
4.0	14.14	13.46	12.77	11.81
4.5	15.87	15.09	14.32	13.23
5.0	17.61	16.75	15.88	14.64
5.5	19.35	18.41	17.46	16.07
6.0	21.09	20.07	19.04	17.40
6.5	22.86	21.76	20.63	18.93
7.0	24.63	23.45	22.23	20.39
7.5	26.41	25.18	23.86	21.85
8.0	28.21	26.91	25.51	23.33
8.5	30.03	28.67	27.18	24.84
9.0	31.87	30.45	28.87	26.36
9.5	33.73	32.27	30.59	27.91
10.0	35.60	34.09	32.34	29.49
10.5	37.52	35.95	34.09	31.07
11.0	39.43	37.82	35.90	32.70
11.5	41.37	39.72	37.72	34.35
12.0	43.33	41.68	39.59	36.03
12.5	45.33	43.65	41.47	37.73
13.0	47.38	45.62	43.40	39.49
13.5		47.67	45.37	41.26
14.0		49.74	47.36	43.07
14.5		51.84	49.39	44.92
15.0		53.94	51.43	46.80
15.5			53.56	48.72
16.0			55.67	50.67
16.5			57.85	52.65
17.0			60.10	54.70
17.5				56.77
18.0				58.87
18.5				61.04
19.0				63.23
19.5				65.48
20.0				67.77

* Subscripts 0 and R refer to tonometer scale readings zero and R.

TABLE 5a

PRESSURE AND RIGIDITY TABLE FOR PAIRED READINGS WITH 5.5- AND 10.0-GM. WEIGHTS[3]

READING WITH 10.0-GM. WEIGHT

READING WITH 5.5 GM. WEIGHT	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0	10.5	11.0	11.5	12.0	12.5	13.0	13.5	14.0	14.5	15.0	15.5	16.0	16.5	17.0	17.5	18.0	18.5	19.0	19.5	20.0
0.0	0840 22	0480 32	0290 38	0178 43	0104 46	0054 49	0019 50																												
0.5	1765 6	0856 18	0496 27	0309 34	0196 39	0122 42	0072 45	0037 46	0011 48																										
1.0			0872 15	0512 24	0324 30	0211 35	0138 38	0088 41	0052 43	0025 44																									
1.5				0883 12	0521 20	0333 27	0222 31	0150 35	0100 37	0064 39	0037 41	0017 42																							
2.0					0889 10	0525 18	0341 24	0231 28	0159 32	0110 34	0074 36	0047 38	0027 39	0012 40																					
2.5						0887 8	0528 15	0346 21	0238 26	0167 29	0117 32	0082 34	0055 35	0036 37	0020 38																				
3.0							0904 6	0536 13	0353 19	0244 23	0173 26	0124 29	0088 31	0063 33	0049 34	0027 35	0015 36																		
3.5								0877 7	0526 12	0350 17	0244 21	0176 24	0127 27	0094 29	0068 31	0048 32	0033 33	0021 34	0011 35																
4.0									0870 5	0523 10	0349 15	0245 19	0177 22	0132 25	0098 27	0073 29	0053 30	0038 31	0026 32	0016 33															
4.5											0517 9	0346 14	0244 18	0180 21	0135 23	0102 25	0076 27	0057 28	0042 29	0030 30	0020 31	0012 32													
5.0												0511 8	0342 12	0246 16	0182 19	0137 21	0104 24	0080 25	0061 26	0046 28	0034 29	0024 29	0016 30												
5.5													0498 7	0341 11	0245 15	0182 18	0138 20	0106 22	0082 24	0063 25	0049 26	0037 27	0027 28	0019 28	0013 29										
6.0														0493 6	0336 10	0242 13	0181 16	0139 19	0107 21	0084 22	0066 24	0051 25	0039 26	0030 26	0022 27	0016 28	0010 28								
6.5															0486 5	0332 9	0240 12	0181 15	0139 17	0109 19	0085 21	0067 22	0053 23	0042 24	0032 25	0025 26	0018 26	0013 27							
7.0																0475 5	0325 8	0237 11	0179 14	0139 16	0109 18	0086 20	0069 21	0055 22	0044 23	0034 24	0027 25	0019 25	0015 26	0010 26					
7.5																		0320 7	0233 10	0178 13	0138 15	0109 17	0086 19	0070 20	0056 21	0045 22	0036 23	0029 23	0022 24	0017 24	0012 25				
8.0																			0313 7	0230 10	0176 12	0137 14	0109 16	0087 18	0071 19	0057 20	0046 21	0037 22	0030 22	0024 23	0019 23	0014 24	0010 24		
8.5																				0307 6	0226 9	0173 11	0136 13	0108 15	0088 17	0071 18	0058 19	0047 20	0039 21	0031 21	0025 22	0020 22	0016 23	0012 23	
9.0																					0300 6	0222 8	0171 11	0135 13	0108 14	0087 16	0072 17	0059 18	0048 19	0040 20	0033 20	0026 21	0021 22	0017 22	0013 22

TABLE 5b

PRESSURE AND RIGIDITY TABLE FOR PAIRED READINGS WITH 7.5- AND 15.0-GM. WEIGHTS[3]

READING WITH 15.0-GM. WEIGHT

READING WITH 7.5 GM. WEIGHT	3.0	3.5	4.0	4.5	5.0	5.5	6.0	6.5	7.0	7.5	8.0	8.5	9.0	9.5	10.0	10.5	11.0	11.5	12.0	12.5	13.0	13.5	14.0	14.5	15.0	15.5	16.0	16.5	17.0	17.5	18.0	18.5	19.0	19.5	20.0
0.0	1870 16	1024 31	0634 43	0414 51	0276 56	0192 60	0118 64	0072 66	0037 68	0011 70																									
0.5		1934 11	1054 25	0657 36	0434 45	0311 50	0203 55	0138 58	0091 61	0056 63	0029 64																								
1.0			2012 8	1083 20	0674 31	0450 38	0311 45	0219 50	0153 53	0106 56	0071 58	0044 59	0023 61																						
1.5					1100 16	0744 25	0460 34	0323 40	0231 45	0166 48	0119 51	0083 53	0056 55	0035 56	0019 57																					
2.0						1253 11	0693 23	0470 30	0331 36	0240 41	0176 44	0129 47	0093 49	0066 51	0045 53	0029 54	0015 55																				
2.5							1127 10	0702 19	0475 27	0338 33	0247 37	0183 41	0137 43	0102 46	0075 48	0054 49	0037 50	0024 51	0013 52																	
3.0								1145 8	0705 16	0480 23	0343 29	0252 34	0189 37	0143 40	0108 43	0082 44	0061 46	0044 47	0031 48	0020 49	0011 50															
3.5									1146 6	0707 14	0481 21	0345 26	0256 31	0194 34	0148 37	0114 39	0087 41	0067 43	0050 44	0037 45	0026 46	0016 49														
4.0											0710 12	0481 18	0347 24	0259 28	0197 32	0152 34	0118 37	0092 39	0071 40	0055 42	0041 43	0031 44	0021 44	0014 45												
4.5												0705 10	0482 16	0347 21	0260 25	0199 29	0155 32	0122 34	0096 36	0075 38	0059 39	0046 40	0035 41	0026 42	0018 43	0012 43									
5.0													0705 8	0480 14	0347 19	0261 23	0201 27	0157 30	0124 32	0099 34	0079 35	0063 37	0049 38	0038 39	0029 40	0022 41	0016 41	0010 42							
5.5														0701 7	0475 12	0345 17	0260 21	0201 24	0159 27	0126 30	0101 32	0082 33	0065 35	0052 36	0042 37	0033 38	0025 39	0019 39	0013 40						
6.0															0694 6	0472 11	0343 15	0259 19	0202 23	0160 26	0127 28	0102 30	0083 32	0068 33	0055 34	0044 35	0035 36	0028 37	0022 37	0016 38	0012 38				
6.5																	0479 9	0346 14	0262 17	0204 21	0161 24	0130 26	0105 28	0086 30	0070 31	0057 32	0047 33	0058 34	0030 35	0024 36	0019 36	0014 37	0010 37		
7.0																		0462 8	0337 13	0257 16	0200 19	0160 22	0129 24	0105 26	0086 28	0071 29	0059 31	0046 32	0040 32	0032 33	0026 34	0021 35	0016 35	0012 36	
7.5																			0458 7	0334 11	0254 15	0199 18	0159 21	0129 23	0106 25	0087 26	0072 28	0060 29	0050 30	0041 31	0034 32	0028 32	0023 33	0018 34	0014 34
8.0																				0453 6	0330 10	0252 14	0198 17	0159 19	0129 21	0106 23	0088 25	0073 26	0061 28	0051 29	0043 29	0036 30	0029 31	0024 32	0020 32
8.5																					0446 6	0326 9	0249 13	0196 15	0158 18	0129 20	0106 22	0086 24	0074 25	0062 26	0062 27	0044 28	0037 29	0031 30	0026 30
9.0																						0440 5	0322 8	0248 12	0194 14	0157 17	0128 19	0106 21	0089 22	0074 24	0063 25	0053 26	0045 27	0058 28	0032 28

TABLE 6[8]

VOLUME OF CORNEAL INDENTATION V_o CU. MM.

Scale Reading	PLUNGER LOAD 5.5 gm.	7.5 gm.	10 gm.	15 gm.
0.0	4.38	3.43	2.73	1.92
0.5	4.96	3.93	3.15	2.24
1.0	5.59	4.47	3.60	2.58
1.5	6.25	5.04	4.09	2.96
2.0	6.95	5.65	4.62	3.37
2.5	7.69	6.30	5.19	3.81
3.0	8.46	6.99	5.78	4.28
3.5	9.27	7.71	6.42	4.79
4.0	10.12	8.48	7.10	5.33
4.5	11.01	9.28	7.81	5.90
5.0	11.94	10.13	8.56	6.51
5.5	12.90	11.01	9.36	7.16
6.0	13.89	11.93	10.19	7.84
6.5	14.94	12.89	11.06	8.55
7.0	16.01	13.89	11.97	9.31
7.5	17.12	14.94	12.92	10.10
8.0	18.27	16.02	13.92	10.93
8.5	19.46	17.15	14.96	11.81
9.0	20.69	18.32	16.04	12.72
9.5	21.95	19.54	17.16	13.67
10.0	23.25	20.79	18.32	14.67
10.5	24.60	22.08	19.53	15.70
11.0	25.97	23.41	20.79	16.78
11.5	27.38	24.78	22.08	17.90
12.0	28.82	26.22	23.43	19.06
12.5	30.32	27.68	24.81	20.26
13.0	31.87	29.17	26.24	21.52
13.5	33.43	30.73	27.73	22.81
14.0	35.03	32.33	29.25	24.15
14.5	36.66	33.97	30.82	25.54
15.0	38.33	35.64	32.42	26.97
15.5	40.06	37.37	34.10	28.45
16.0	41.81	39.13	35.79	29.97
16.5	43.62	40.94	37.55	31.54
17.0	45.45	42.79	39.38	33.17
17.5	47.32	44.71	41.23	34.84
18.0	49.19	46.67	43.11	36.54
18.5	51.17	48.69	45.07	38.32
19.0	53.13	50.71	47.05	40.13
19.5	55.20	52.77	49.11	42.01
20.0	57.23	54.91	51.21	43.93

TABLE 7[8]

INTRAOCULAR PRESSURE DURING TONOMETRY P_t MM. HG.

Scale Reading	PLUNGER LOAD 5.5 gm.	7.5 gm.	10 gm.	15 gm.
0.0	51.40	70.09	93.46	140.19
0.5	48.29	65.85	87.80	131.69
1.0	45.53	62.09	82.78	124.17
1.5	43.07	58.73	78.31	117.46
2.0	40.86	55.72	74.29	111.44
2.5	38.87	53.00	70.67	106.01
3.0	37.06	50.54	67.39	101.08
3.5	35.42	48.29	64.39	96.59
4.0	33.91	46.24	61.65	92.48
4.5	32.53	44.35	59.14	88.70
5.0	31.25	42.61	56.82	85.23
5.5	30.07	41.01	54.67	82.01
6.0	28.98	39.52	52.69	79.03
6.5	27.96	38.13	50.84	76.26
7.0	27.01	36.84	49.12	73.67
7.5	26.13	35.63	47.51	71.26
8.0	25.30	34.50	46.00	69.00
8.5	24.52	33.44	44.58	66.87
9.0	23.79	32.44	43.25	64.88
9.5	23.10	31.50	42.00	63.00
10.0	22.45	30.61	40.82	61.22
10.5	21.83	29.77	39.70	59.55
11.0	21.25	28.98	38.64	57.96
11.5	20.70	28.23	37.64	56.45
12.0	20.18	27.51	36.68	55.03
12.5	19.68	26.83	35.78	53.67
13.0	19.20	26.19	34.92	52.37
13.5	18.75	25.57	34.09	51.14
14.0	18.32	24.98	33.31	49.97
14.5	17.91	24.42	32.56	48.84
15.0	17.52	23.89	31.85	47.77
15.5	17.14	23.37	31.16	46.74
16.0	16.78	22.88	30.51	45.76
16.5	16.43	22.41	29.88	44.82
17.0	16.10	21.96	29.27	43.91
17.5	15.78	21.52	28.69	43.04
18.0	15.48	21.10	28.14	42.21
18.5	15.18	20.70	27.60	41.40
19.0	14.90	20.31	27.09	40.63
19.5	14.62	19.94	26.59	39.88
20.0	14.36	19.58	26.11	39.16

$P_{o_{corr}}$ is the intraocular pressure of the undisturbed eye as estimated either from scleral rigidity measurements with the Schiøtz tonometer (Table 5 or nomogram) or as measured with the applanation tonometer.

The duration of the tonogram is assumed to be four minutes.*

These values are then substituted into the above expression and arithmetically simplified.

* The steps in arriving at this expression are directed to removing the average coefficient of scleral rigidity (E_{av}) employed by Grant in determining volume loss during tonography ($^{\Delta}V_G$) and introducing the correct scleral rigidity (E_{corr}) and consequently the correct volume loss ($^{\Delta}V_{corr}$).

Grant's tonographic equation may be written

$$C = \frac{^{\Delta}V_G}{t(P_{t_{av}} - P_{o_{av}} - {}^{\omega}P_v)}$$

$^{\Delta}V_G = {}^{\Delta}V_o + {}^{\Delta}V_{s_{av}}$ in which $^{\Delta}V_o$ is the change in corneal indentation volume during tonography and $^{\Delta}V_{s_{av}}$ is the loss of volume due to "scleral shrinkage" if the eye has an average scleral rigidity (E_{av}).

(Footnote continued on next page)

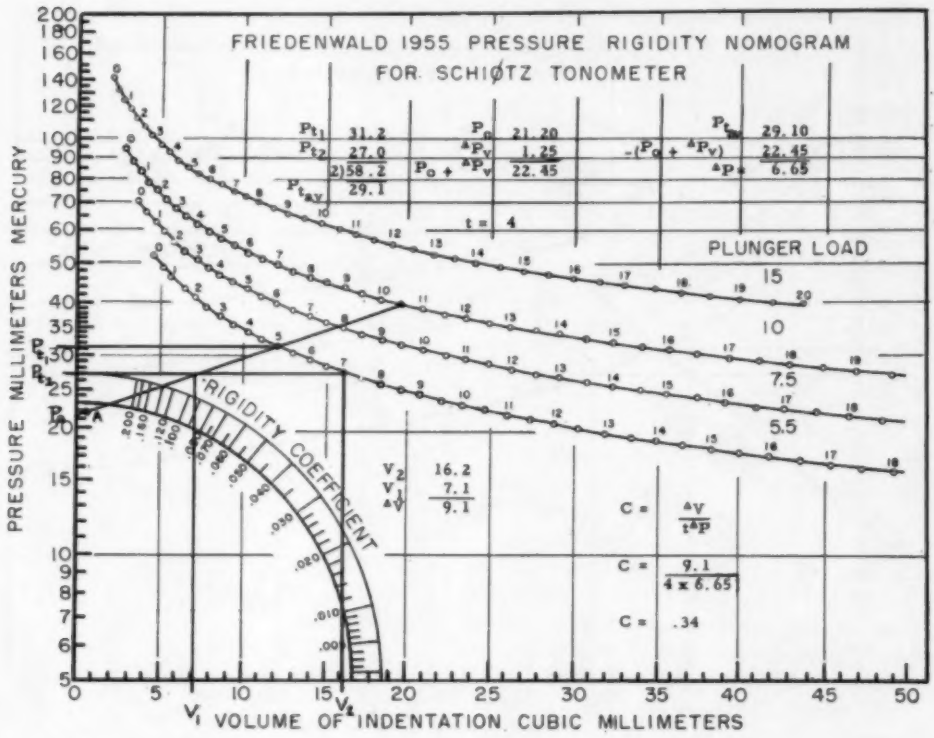


Fig. 2 (Moses and Becker). Graphic representation of tonogram with 5.5-gm. weight where initial scale reading was 5.0 and four-minute reading was 7.0, assuming a low value for the scleral rigidity coefficient (E = 0.0137).

$$\Delta V_{e_{av}} = \frac{1}{E_{av}} \log \frac{P_{t_1}}{P_{t_2}}$$

$$\Delta V_{e_{corr}} = \frac{E_{av}}{E_{corr}} \frac{1}{E_{av}} \log \frac{P_{t_1}}{P_{t_2}} = \Delta V_{e_{av}} \frac{E_{av}}{E_{corr}}$$

$$\Delta V_{corr} = \Delta V_e + \Delta V_{e_{av}} \frac{E_{av}}{E_{corr}}$$

Since tables of $\Delta V_{e_{av}}$ are not available this value may be obtained from

$$\Delta V_{e_{av}} = \Delta V_0 - \Delta V_e, \text{ so}$$

$$\Delta V_{corr} = \Delta V_e + (\Delta V_0 - \Delta V_e) \frac{E_{av}}{E_{corr}} \text{ and}$$

$$C = \frac{\Delta V_e + (\Delta V_0 - \Delta V_e) \frac{E_{av}}{E_{corr}}}{t(P_{t_{av}} - P_{o_{corr}} - \Delta P_v)}$$

If $E_{av} = 0.0215$, $t = 4$, and $\Delta P_v = 1.25$ the expression simplifies to that given above.

Example 1 (a low scleral rigidity corresponding to that used in fig. 2).

Data:

$R_{10} = 10.5$

$R_{5.5_1} = 5.0$

$R_{5.5_2} = 7.0$

A = 22 mm. Hg (applanation)

From table number

Va E = 0.0137

Va $P_{corr} = 21$

IV $\Delta V_0 7.0_{5.5} = 24.63$

IV $-\Delta V_0 5.0_{5.5} = -17.61$

$\Delta V_0 = 7.02$

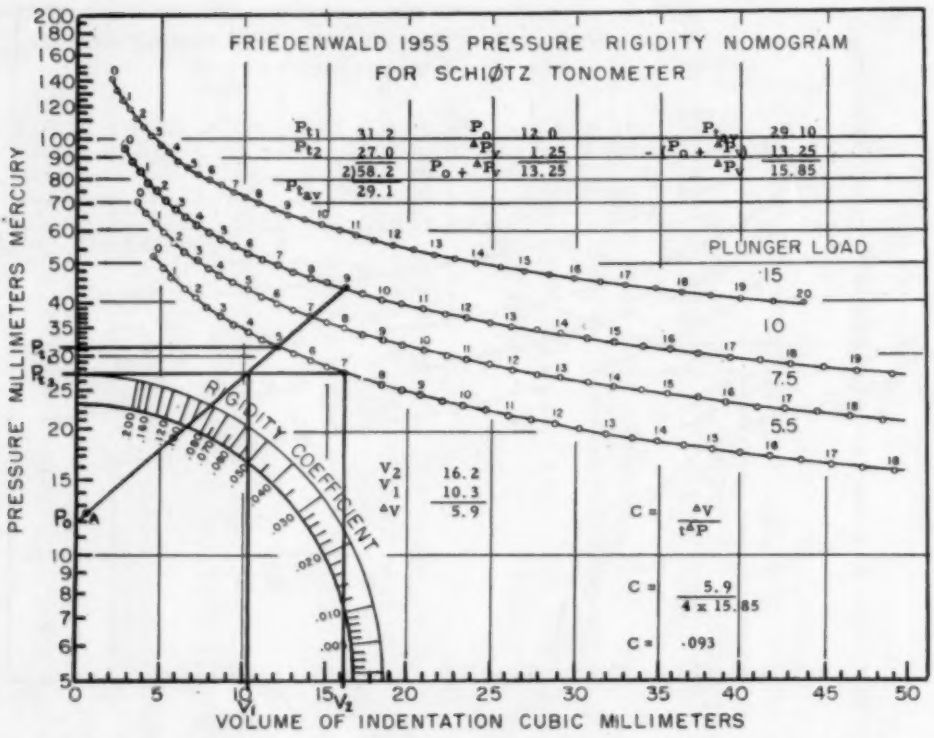


Fig. 3 (Moses and Becker). Graphic representation of tonogram with 5.5-gm. weight where initial scale reading was 5.0 and four-minute reading was 7.0, assuming a high value for the scleral rigidity coefficient (E = 0.0342).

VI $V_e 7.0_{5.5} = 16.01$

VI $-V_c 5.0_{5.5} = -11.94$

$\Delta V_e = 4.07$

VII $P_{t_1} 5.0_{5.5} = 31.25$

VII $P_{t_2} 7.0_{5.5} = 27.01$

$2) \quad 58.26$

$P_{t_{av}} = 29.13$

$$C = \frac{0.0215 \Delta V_G + (E - 0.0215) \Delta V_e}{4E(P_{t_{av}} - P_{o_{corr}} - 1.25)}$$

$$= \frac{0.0215 \times 7.02 + (0.0137 - 0.0215) \times 4.07^*}{4 \times .0137 \times (29.13 - 21 - 1.25)}$$

$C = 0.32$

Example 2 (a high scleral rigidity corresponding to that used in fig. 3).

Data:

$R_{10} = 9.0$

$R_{5.5_1} = 5.0$

$R_{5.5_2} = 7.0$

A = 12.5 mm. Hg (applanation)

* When scleral rigidity is *less* than average $0.0137 - 0.0215 = -0.0078$) the second part of the numerator has a negative value and must be *subtracted* from the first part.

TABLE 8

TONOGRAPHIC RESULTS AS OBTAINED BY DIFFERENT METHODS OF CALCULATION
$R_{5.5_1}=5.0$, $R_{5.5_2}=7.0$

A	R_{10}		P_o	C	P_o/C
17.5	9.75	Average rigidity (Table 1)	17.0	0.17	100
		Graph (Figure 1)	17.0	0.17	100
22.0	10.50	Graph (Figure 2)	21.2	0.34	62
		Tabular (Example 1)	21.0	0.32	66
12.5	9.0	Graph (Figure 3)	12.0	0.093	129
		Tabular (Example 2)	12.0	0.093	129

A = applanation reading (mm. Hg).
$R_{5.5}$ = scale reading Schiøtz tonometer with 5.5 gm. weight.
R_{10} = scale reading Schiøtz tonometer with 10-gm. weight.
P_o = intraocular pressure corrected for scleral rigidity.
C = facility of outflow corrected for scleral rigidity.

From table number

Va $E = 0.0342$

Va $P_{o_{corr}} = 12$

IV $\quad {}^{\Delta}V_G 7.0_{5.5} = 24.63$

IV $\quad -{}^{\Delta}V_G 5.0_{5.5} = -17.61$

${}^{\Delta}V_G = 7.02$

VI $\quad V_c 7.0_{5.5} = 16.01$

VI $\quad -V_c 5.0_{5.5} = -11.94$

${}^{\Delta}V_c = 4.07$

VII $\quad P_{t_1} 5.0_{5.5} = 31.25$

VII $\quad P_{t_2} 7.0_{5.5} = \dfrac{27.01}{2)\,58.26}$

$P_{t_{av}} = 29.13$

$$C = \frac{0.0215\,{}^{\Delta}V_G + (E - 0.0215)\,{}^{\Delta}V_c}{4E(P_{t_{av}} - P_{o_{corr}} - 1.25)}$$

$$= \frac{0.0215 \times 7.02 + (0.0342 - 0.0215)4.07}{4 \times .0342(29.13 - 12 - 1.25)}$$

$C = 0.093$

Discussion

The results of the above methods of computing a given tonogram with different scleral rigidity values and by different methods are assembled in Table 8.

It is seen that there is reasonably good agreement between the results obtained by the two methods.

It is also seen that while a correction for scleral rigidity changes the P_o and C values and the interpretation of the mechanism of glaucoma involved, it does not usually remove a borderline tracing from the group of eyes where glaucoma is suspected. Thus, in the case of average scleral rigidity, $C=0.17$ is a borderline value. With the correction for an assumed low scleral rigidity C rises to 0.34 but P_o also rises to 21.2, a value high enough to arouse suspicion of hypersecretion glaucoma in the face of such a good value for C. When the tonogram is corrected for an assumed high scleral rigidity, P_o becomes definitely normal, but $C=0.093$ and one must conclude that this is an eye with poor facility of outflow, but in a hyposecretory phase.

Summary and Conclusions

Simplified tables for calculating coefficient of facility of outflow from tonographic data are presented.

Two methods are offered for the correction of tonographic data for individual variations in coefficient of scleral rigidity.

640 South Kingshighway (*10*).

References

1. Grant, W. M.: Clinical measurement of aqueous outflow. Arch. Ophth. **46**:113, 1951.
2. Becker, B., and Friedenwald, J. S.: Clinical aqueous outflow. Arch. Ophth., **50**:557, 1953.
3. Friedenwald, J. S.: Contribution to the theory and practice of tonometry. Am. J. Ophth., **20**:985, 1937.
4. Ballantine, E. J.: Tonography and aqueous humor dynamics: The effect of variation in scleral rigidity on the apparent facility of aqueous drainage as calculated from tonographic measurements. Acta. XVII Conc. Ophth. (1954) **3**:1516, 1955.
5. Friedenwald, J. S.: Tonometer calibration: An attempt to remove discrepancies found in 1954 calibration for Schiøtz tonometers. Tr. Am. Acad. Ophth., **61**:108, 1957.
6. Linnér, E.: Episcleral venous pressure during tonography. Acta. XVII Conc. Ophth., 1954, **3**:1532, 1955.
7. Moses, R., and Hahn, K.: The effect of the tonometer footplate hole on scale reading. Arch. Ophth., in press.
8. Moses, R.: Unpublished observations, 1956.
9. Goldmann, H., and Schmidt, T.: Der Rigiditätskoeffizient. Ophthalmologica, **133**:330, 1957.

ORBITONOMETRY IN UNILATERAL EXOPHTHALMOS*

JOHN A. DYER, M.D., AND JOHN W. HENDERSON, M.D.
Rochester, Minnesota

Since the introduction of the orbitonometer by Copper[1] and the publication of his monograph on its clinical application in Graves' disease, acromegaly, orbital inflammation, and tumors, little additional information concerning the use of this instrument in exophthalmos has been obtained. Copper thought that the orbitonometer had certain clinical applications. Because of lack of confirmation of his work with regard to orbital tumors and inflammation, the present study was undertaken to determine if the orbitonometer is useful as a routine clinical aid in the diagnosis and differentiation of orbital space-occupying lesions that produce unilateral proptosis.

Comparison of the resistance to retrodisplacement of the globe in exophthalmos with that of normal eyes may make it possible to reach a more definite conclusion as to the effectiveness of the instrument in the diagnosis of the underlying mechanism producing exophthalmos.

General background

As early as 1868, Graefe[2] noted that one of the first symptoms of orbital extension of an intraocular tumor was, in addition to proptosis, increased resistance when an attempt was made to force the eye against the posterior orbital wall. Numerous endeavors to develop some mechanical means of measuring increased orbital resistance in unilateral exophthalmos were made by Langenhan,[3] Gutmann,[4,5] Plegge,[6] and Georg,[7] but the technical and practical disadvantages of these instruments did not permit their general clinical application.

Copper, in 1948, invented an instrument that proved to be more easily adapted to routine study. He coined the word "orbitonometer" for it and described the method of measuring orbital resistance with it as "orbitonometry." Means and Stanbury,[8] Galindez Iglesias,[9] and Grossmann and Burns[10] found this instrument to be satisfactory in measuring orbital resistance in the exophthalmos of Graves' disease. Kearns and associates,[11] in a recent study, utilized the orbitonometer in cases of exophthalmos as-

* From the Section of Ophthalmology, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota. Abridgment of thesis submitted by Dr. Dyer to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Ophthalmology.

sociated with Graves' disease. They found that use of the instrument could not differentiate exophthalmos into thyrotoxic and thyrotropic types but noted that orbital "tension" (resistance of the orbital tissues to retrodisplacement of the globe) was increased in proportion to the degree of exophthalmos. The accurate determination of the degree of exophthalmos made possible with the orbitonometer was found to be of value, since the Hertel exophthalmometer was subject to a certain degree of error due to parallax.

In addition to its use in exophthalmos of thyroidal origin, Copper applied his instrument in cases in which inflammatory conditions of the orbit or tumors were producing unilateral proptosis. He concluded that orbitonometry allowed early recognition of inflammation of the accessory sinuses with orbital involvement by the detection of increased orbital "tension" and that neoplasms and pseudotumors could be diagnosed by the same sign even in the absence of proptosis. He considered that the last two entities could not be differentiated by a single measurement and that orbitonometry did not diagnose the nature of a tumor but that it was capable of proving, in an early stage, the presence of abnormal density and that repeated measurements were valuable in noting whether the process was progressive or stationary.

Faldi[12] reported use of the orbitonometer in six cases of exophthalmos caused by orbital lesions, finding a striking difference between the measurements in exophthalmos resulting from vascular conditions and neoplastic causes.

The orbitonometer

The instrument is manufactured by C. W. Dixey and Son, Ltd., London, England. It consists of three parts, namely the dynamometer, the supporting bridge, and the contact shells (fig. 1).

The aluminum dynamometer is cylindric and approximately 25 cm. in length, with a spring enclosed in the handle. The stem is

Fig. 1 (Dyer and Henderson). The orbitonometer.

calibrated at its upper portion so that it measures from 100 to 400 gm. as pressure is applied to the handle, the lower edge of the latter indicating the amount of force applied. The distal portion of the stem is calibrated in millimeters, and the degree of exophthalmos is noted here as it passes through the supporting bridge (fig. 2). The instrument is constructed so that the height of the cornea above the lateral orbital rim may be read directly from this millimeter scale, thus giving an accurate measurement of the degree of prominence of the globe.

The supporting bridge, which can be adjusted to the patient's intraorbital width and locked into position by set screws, is supported by notched flanges on the legs of the instrument that fit snugly on the patient's lateral orbital rims. The nose support is adjustable and serves to prevent vertical displacement of the bridge rather than to support the weight of the instrument. An elastic and adjustable headband holds the bridge firmly in position.

The contact shells, which are plastic and resemble the usual contact lenses, are of three sizes, with varying corneal diameters and radii of curvature. The outer edge of the shell is fitted with a receptacle into which the stem of the dynamometer rests snugly.

Use of the instrument

With the patient reclining, a drop of a 0.5-percent solution of tetracaine (Ponto-

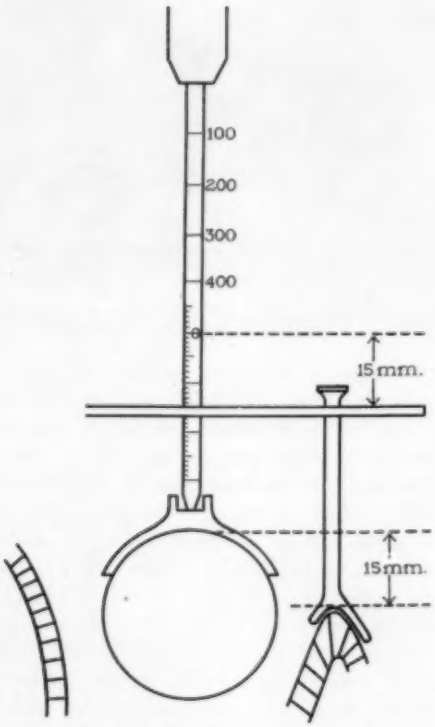


Fig. 2 (Dyer and Henderson). The calibrations on the stem of the orbitonometer.

caine) hydrochloride is instilled in each eye. While the effect is awaited, the supporting bridge is adjusted on the orbital rims. The elastic headband is then fitted, with sufficient tension to assure firm support. The nosepiece is positioned and lowered to the bridge of the nose, care being taken that it does not alter the distance of the horizontal bar from the patient's eyes. The instrument then will remain in correct position.

After a second instillation of Pontocaine, the contact shell, which previously has been cleansed in alcohol, rinsed in water and dried, is inserted between the lids in the manner of a prosthesis. The stem of the dynamometer is inserted through the slit in the horizontal bar into the recess of the shell, and the patient is directed to look, with the uncovered eye, through the slit of the horizontal bar toward the examiner's fingers in order to keep the eyes vertical and stabilized (fig. 3). With the dynamometer held vertically, an initial reading without pressure is made, which gives the height of the cornea above the orbital rim in millimeters (zero reading). Pressures of 100, 200, and 300 gm. are applied in turn, permitting a few seconds between readings for stabilization of the globe, and the distance in millimeters again is read from the scale for each new weight. The pressure should be released slowly after each measurement. The opposite eye is then measured similarly.

Method of recording

In order to be systematic in recording the results, the methods of Copper and Kearns and co-workers were utilized in this study. The amount of retrodisplacement of the globe by a known pressure was measured and recorded.

E and Y values were used, the E value representing the direct reading of the position of the globe with a given pressure (absolute value), and the Y value representing the millimeters of retrodisplacement (relative value).

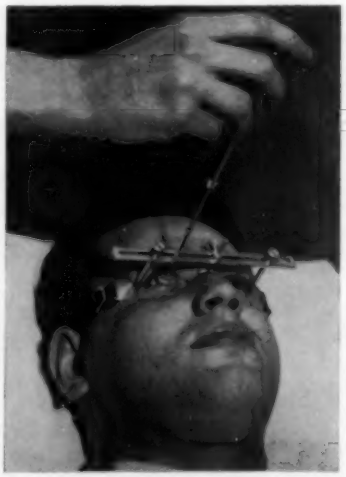


Fig. 3 (Dyer and Henderson). The orbitonometer in position.

E_0 is the original position of the cornea in relation to the orbital rim; in other words, the handle is at the zero mark (no pressure applied). E_{100}, E_{200}, and E_{300} refer to the new corneal positions as pressures of 100, 200, and 300 gm., respectively, are applied. Y_{100} refers to the amount of posterior displacement of the globe in millimeters with the 100-gm. pressure; it equals E_{100} minus E_0. Y_{200} refers to the amount of posterior displacement with the 200-gm. pressure (E_{200} minus E_0), and Y_{300} is equal to E_{300} minus E_0.

Normal values

The values recorded as normal by Copper, who measured 120 normal eyes, have been substantiated by Kearns and associates, who measured a total of 28 normal eyes. The findings for the normal eye of each of the 36 patients in our present study bear out their figures (table 1 and fig. 4). As noted in Figure 4, the curve for normal eyes is gradual, flattening out somewhat with the use of greater pressure.

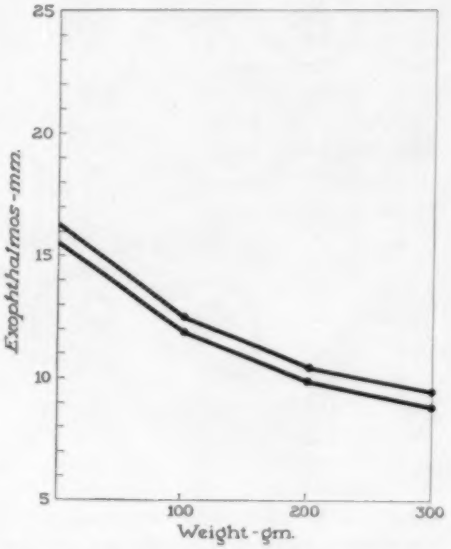


Fig. 4 (Dyer and Henderson). Average orbitonometric findings for normal eyes. The upper curve utilizes the data of Copper, whereas the lower curve is based on data of Kearns and associates. The values plotted are E values.

Copper used pressures up to 400 gm. in his studies, but it was our personal experience, as well as that of Kearns and associates and Galindez Iglesias, that 300 gm. of pressure was the most that could be tolerated by the patient. Since undue manipulation of the globe might produce further damage, patients were omitted from the series if they had ocular disease accompanied by hemorrhagic manifestations, retinal detachment, acute inflammations, glaucoma or cardiac arrhythmias, or if they had pain in and around the orbit such as to preclude application of the instrument.

Mechanism of exophthalmos

According to Kearns and co-workers,[11] the most probable mechanisms of exophthalmos in Graves' disease are an increase in the amount of orbital fat, edema of the soft

TABLE 1
MEAN NORMAL ORBITONOMETRIC MEASUREMENTS

Authors	Posterior Displacement (mm.)		
	Y_{100}	Y_{200}	Y_{300}
Copper[1]	3.6 (0.8)*	5.8 (0.9)	6.8 (1.0)
Kearns and associates[11]	3.6 (0.8)	5.6 (0.9)	6.7 (1.0)
Dyer and co-workers (present study)	3.7 (0.7)	5.8 (1.0)	6.8 (1.2)

* Numbers in parentheses are standard deviations.

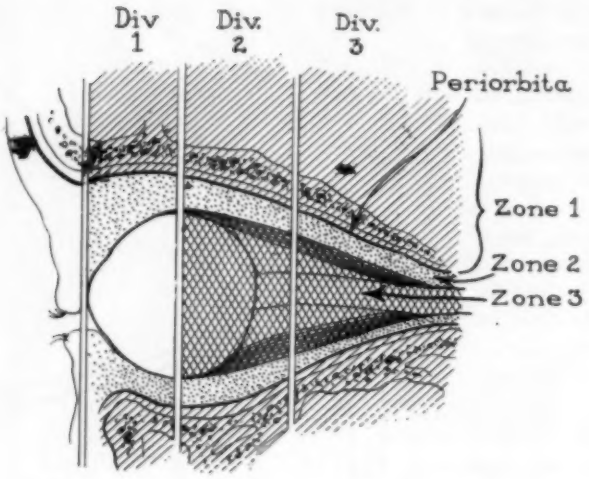


Fig. 5 (Dyer and Henderson). Anatomic division of the orbit.

tissues of the orbit, and enlargement of the extraocular muscles. Lymphocytic infiltration of the orbital soft tissues and muscles also was noted.

Benedict[13] described the orbit as being divided anatomically into three divisions, namely anterior, middle, and posterior, as well as three zones, the subperiosteal zone, the zone between the periosteum and the muscle cone, and the zone within the muscle cone (fig. 5). He considered that these subdivisions simplified the problem of surgical intervention and greatly facilitated the diagnosis of orbital diseases.

Space-occupying lesions of the first zone may be found anywhere within the orbit. Lesions in the first division produce less proptosis of the globe than do those situated more posteriorly, but lateral or vertical displacement may be more pronounced. Within the second zone are noted the greatest number and variety of space-occupying lesions of vascular and neoplastic origin. Proptosis is characteristic of tumors of this zone located in the posterior division (division 3). Most tumors in this location are primary, with proptosis, displacement of the globe, and swelling of the eyelids being the most characteristic signs, although these do not always denote adequately the size, shape, and position of the tumor. Space-occupying lesions within the muscle cone (zone 3) are situated in the middle and posterior divisions. The distinguishing feature of lesions here is proptosis that is straight forward, without lateral displacement, plus visual loss, choked disc, and wrinkling of the retina from pressure by the tumor on the globe.

Copper also commented on the mechanism of displacement of the globe with orbital space-occupying lesions, stating that the greatest degree of deformity of the tissues occurred from lesions within the muscle cone, since local increased density in that location would produce the greatest limitation of backward displacement of the globe. Anatomically, the globe lies in greater proximity to the lateral orbital wall than to the other walls, so that pressure directly on the globe forces it toward this wall and may bring it into direct contact if sufficient pressure is applied; this results in the eye being displaced posteronasally. Because of this anatomic arrangement, a temporally situated lesion will tend to limit displacement more than a nasally situated one. In this regard, it is interesting to note that Kearns and Wagener,[14] in an article on meningiomas of

the sphenoidal ridge, found that en plaque (pterional) tumors produced exophthalmos as the earliest symptom; the proptosis was rather pronounced, averaging 5.9 mm., and was straight forward or forward and downward, with great resistance to retrodisplacement of the globe.

In Copper's opinion, the degree of posterior displacement of the globe under an applied force depends on two factors, namely the volume of the retrocapsular space, determined by the relationship of ocular size to the size of orbital contents, and the orbital "tension" produced by the consistency of the tissue, the tone of the muscle, and the degree of expulsion of fluid. He considered that muscular tone had a positive effect on orbital tension in that it is active in all phases of measurements, that a localized area of density situated inside the muscle cone or diffuse infiltration inhibited retrodisplacement of the globe in the middle and last stages of a measurement, with resultant flattening of the curve, that diffuse mild edema resulted in flattening of the curve, with little effect on total displacement, and that expulsion of fluid occurred predominantly in the beginning stage of measurement with the 100-gm. and 200-gm. pressures. He noted that overlapping of these factors may occur. Therefore, it appeared probable that a solid carcinoma would produce the greatest degree of resistance, whereas a vascular tumor, such as an angioma, would cause the least.

Present study

The series in the present study consisted of 36 instances of unilateral exophthalmos seen at the Mayo Clinic and occurring in the following conditions: Graves' disease, six cases; pseudotumor, five cases; meningioma, four cases; metastatic carcinoma, three cases; hemangioma, three cases; undetermined, two cases; mucocele, two cases; lymphoma, two cases; aneurysm of the internal carotid artery, two cases; and one case each of hemangio-endothelioma, eosinophilic granuloma, astrocytoma of the optic nerve, benign fibrodysplasia of the skull, Paget's disease of the skull, osteoma of the orbit, and juvenile fibromas of the nasopharynx. The term "tumor" in this study denotes any space-occupying lesion, whether carcinoma, an inflammatory mass, or diffuse infiltration or edema of tissue.

The patients were selected as they reported to the Section of Ophthalmology with complaints of unilateral proptosis. In all but three instances, orbitonometry was done before either a clinical or pathologic diagnosis had been made in order that the study might be performed as impartially as possible. Only one measurement was made in each case, which allowed appraisal of the diagnostic value of routine orbitonometry. In only one instance was the intraocular pressure increased in the exophthalmic eye; this was an eye that had been blind for a long time after extension of a meningioma into the orbit. Every exophthalmic eye in this series had reduced sensitiveness to compression by the orbitonometer. Compression of the normal eye often resulted in considerable discomfort to the patient in the absence of pain on compression of the other eye. Explanation of the procedure to the patient invariably resulted in better cooperation, lessening the concern of the patient regarding the instrumentation.

It became apparent early in the study that we would be unable to differentiate one type of orbital space-occupying lesion from another simply by means of the orbitonometer; for example, we found increased resistance to retrodisplacement of the globe in the presence of a certain type of tumor and equal resistance with different types of tumor or much less resistance with the same type of tumor that was less advanced or situated in another part of the orbit. This confirms Copper's conclusions as to differentiation of specific types of lesions by orbitonometry alone.

Study of the E values indicated that the patients could be divided into three groups based on the degree of resistance to retrodisplacement of the globe and the anatomic location of the tumor.

Group 1 (10 cases) included patients with maximal degrees of increased resistance to retrodisplacement of the globe; *group 2* (nine cases) comprised those with increased resistance but to a lesser degree than group 1; *group 3* (17 cases) included those in whom the values for posterior displacement resembled those of normal eyes even in the presence of proptosis. After this division, it became apparent that the first and second groups were made up of patients who had tumors located within the posterior two thirds of the orbit and also two patients who had Graves' disease with apparently dense infiltration of the orbital tissue and edema. In the third group, the tumors were anterior in the orbit, outside the orbit posteriorly, or within the orbit but sufficiently small so as not to influence retrodisplacement. Several patients with Graves' disease also were placed in this group; these patients had less tissue changes and, therefore, less resistance to posterior displacement of the globe than did those with Graves' disease in the second group.

The orbitonometric readings and the diagnosis in these three groups are given in Tables 2, 3, and 4.

These data revealed that some factors were common to both groups 1 and 2: (1) the lesions were situated in the middle and posterior divisions of the orbit and were within any of the three zones; (2) the various causes of proptosis were multiple; (3) the degree of exophthalmos was considerably varied, being extremely pronounced in some cases and minimal in others. It appeared initially that these similarities were sufficient to allow these two groups to be considered together as one large group but, as will be shown later, the variation in Y values did not justify such combination.

Study of group 3 also indicated several important factors: (1) the tumors were located in the anterior division of the orbit or outside the orbit posteriorly, or the lesions inside the orbit were too small to produce significant increase in resistance to posterior displacement of the globe; (2) the degree of exophthalmos was variable but was less in most cases than in groups 1 and 2; (3) the Y values were rather consistent and resembled those of the opposite normal eye; (4) the shape of the curve of retrodisplacement resembled the normal average curve in each instance.

Although one must bear in mind the fact that, in clinical orbitonometry, the exophthalmic eye is compared to the opposite normal eye in each case, the normal values are sufficiently consistent to warrant the use of the mean normal values for comparison with the values of the other groups.

In view of the fact that the E values were so variable, a study of the Y values yielded information that was more readily assessed. As noted in Tables 2, 3, and 4, the Y values were essentially consistent in each group, so that use of the mean Y values appeared adequate (table 5). Study of Table 5 confirms the observation that patients in group 1 show the greatest resistance to retrodisplacement, those in group 2 show a lessened resistance to posterior displacement, and those in group 3 have values resembling normal; thus, the last group shows much less resistance to retrodisplacement and is distinctly different from the other two groups.

Figure 6 represents the slopes of the curves for the mean Y values in each group as compared to normal. No attempt was made to indicate the millimeters of exophthalmos above normal but only the significance of the slope of the curve of retrodisplacement in each instance. The flattened slope in group 1 indicates definite resistance; the curve for group 2 also is flattened but less so than that for group 1 and yet more so

TABLE 2

ORBITONOMETRIC MEASUREMENTS IN 10 CASES OF UNILATERAL EXOPHTHALMOS: GROUP 1

Case	Position of Eye (mm.)								Posterior Displacement (mm.)						Diagnosis†
	E_0		E_{100}		E_{200}		E_{300}		Y_{100}		Y_{200}		Y_{300}		
	Ex.*	N.*	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	
1	24.0	20.5	22.5	18.0	21.5	16.5	21.0	15.5	1.5	2.5	2.5	4.0	3.0	5.0	Pseudotumor—floor and apex of orbit
2	17.5	12.5	16.0	9.5	14.5	8.0	13.5	7.0	1.5	3.0	3.0	4.5	4.0	5.5	Cavernous hemangioma—slightly anterior and medial to optic nerve
3	23.0	13.0	21.5	9.0	20.0	6.0	19.0	4.5	1.5	4.0	3.0	7.0	4.0	8.5	En plaque meningioma
4	16.5	11.5	15.0	8.5	13.5	7.0	12.0	6.0	1.5	3.0	3.0	4.5	4.5	5.5	Erosion of optic foramen—aneurysm?
5	23.0	19.0	21.0	15.0	20.0	12.0	19.0	10.5	2.0	4.0	3.0	7.0	4.0	8.5	Pseudotumor—upper nasal orbit to apex
6	21.0	13.0	19.0	9.0	18.0	6.5	17.0	5.0	2.0	4.0	3.0	6.5	4.0	8.0	Lymphoma—upper and lower temporal orbit
7	23.5	14.0	21.0	10.0	20.0	7.5	19.0	6.5	2.5	4.0	3.5	6.5	4.5	7.5	Hemangioma—upper inner orbit to apex
8	26.0	15.0	24.0	12.0	22.5	10.5	21.5	9.5	2.0	3.0	3.5	4.5	4.5	5.5	Meningioma—right frontal canal through foramen into muscle cone
9	22.0	11.5	20.0	7.5	18.5	6.0	17.5	5.0	2.0	4.0	3.5	5.5	4.5	6.5	Metastatic carcinoma from prostate
10	16.0	13.0	13.5	10.0	12.5	9.0	11.5	8.0	2.5	3.0	3.5	4.0	4.5	5.0	Paget's disease of skull

* Ex. = exophthalmic eye; N = normal eye.
† Surgical and pathologic diagnosis was made in all but Cases 9 and 10, in which operation was not done.

TABLE 3

ORBITONOMETRIC MEASUREMENTS IN NINE CASES OF UNILATERAL EXOPHTHALMOS: GROUP 2

Case	Position of Eye (mm.)								Posterior Displacement (mm.)						Diagnosis†
	E_0		E_{100}		E_{200}		E_{300}		Y_{100}		Y_{200}		Y_{300}		
	Ex.*	N.*	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	
11	25.0	11.0	22.0	7.0	21.0	6.0	20.0	5.0	3.0	4.0	4.0	5.0	5.0	6.0	Melanoma (?)—posterior, superior and lateral to second cranial nerve
12	24.0	12.0	21.5	8.0	20.0	6.0	18.5	5.0	2.5	4.0	4.0	6.0	5.5	7.0	Eosinophilic granuloma—retrobulbar and temporal
13	18.0	17.0	15.5	12.0	13.5	10.0	13.0	9.0	2.5	5.0	4.5	7.0	5.5	8.0	Pseudotumor—floor of orbit
14	17.0	13.0	15.0	9.0	13.0	7.5	12.0	5.5	2.0	4.0	4.0	5.5	5.0	6.5	Metastatic carcinoma—primary gastrointestinal tract (?)
15	17.0	12.5	15.0	9.0	13.0	8.0	12.0	7.0	2.0	3.5	4.0	4.5	5.0	5.5	Astrocytoma—optic nerve (orbital) into foramen
16	19.0	13.0	17.0	10.0	15.0	8.0	14.0	7.0	2.0	3.0	4.0	5.0	5.0	6.0	Graves' disease
17	23.0	19.5	21.0	17.0	19.0	15.0	17.5	14.0	2.0	2.5	4.0	4.5	5.5	5.5	Lymphosarcoma (biopsy)
18	24.0	13.0	22.0	9.0	20.0	7.0	18.5	6.5	2.0	4.0	4.0	6.0	5.5	6.5	Pyocele—left ethmoid
19	19.5	16.0	17.0	11.0	15.0	8.0	13.5	7.0	2.5	5.0	4.5	8.0	6.5	9.0	Graves' disease

* Ex. = exophthalmic eye; N = normal eye.

† Surgical and pathologic diagnosis was made in all but Cases 14, 16 and 19, in which operation was not done.

TABLE 4

ORBITONOMETRIC MEASUREMENTS IN 17 CASES OF UNILATERAL EXOPHTHALMOS: GROUP 3

Case	Position of Eye (mm.)								Posterior Displacement (mm.)						Diagnosis
	E_0		E_{100}		E_{200}		E_{300}		Y_{100}		Y_{200}		Y_{300}		
	Ex.*	N.*	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	Ex.	N.	
20	26.5	22.0	24.0	19.5	21.5	16.0	20.0	14.5	2.5	3.0	5.0	6.5	6.5	8.0	†Aneurysm—right carotid
21	16.0	13.5	13.0	9.5	11.0	7.5	10.0	7.0	3.0	4.0	5.0	6.0	6.0	6.5	Metastatic carcinoma—breast?
22	15.0	11.0	12.0	7.5	10.0	5.5	8.5	5.0	3.0	3.5	5.0	5.5	6.5	6.0	Pseudotumor (?)
23	17.5	16.5	14.0	12.5	13.0	11.0	12.0	10.0	3.5	4.0	4.5	5.5	5.5	6.5	†Pseudotumor—lacrimal gland and fat
24	33.0	15.0	29.0	11.0	28.0	9.0	27.0	7.5	4.0	4.0	5.0	6.0	6.0	7.5	Benign fibrodysplasia—skull
25	10.0	9.0	6.0	5.0	5.0	4.0	4.5	3.5	4.0	4.0	5.0	5.0	5.5	5.5	†Osteoma—supraorbital, with edematous intra-orbital muscles
26	13.0	10.0	9.5	6.5	7.0	5.0	6.0	4.5	3.5	3.5	6.0	5.0	7.0	5.5	†Multiple juvenile fibromas—nasopharynx
27	19.0	15.0	16.0	10.0	13.5	8.0	13.0	7.0	3.0	5.0	5.5	7.0	6.0	8.0	†Meningioma—sellar, with optic-nerve compression
28	19.0	16.0	16.0	13.0	13.0	10.0	12.0	9.0	3.0	3.0	6.0	6.0	7.0	7.0	Graves' disease
29	21.0	19.0	17.5	16.0	14.5	14.0	13.0	13.0	3.5	3.0	6.5	5.0	8.0	6.0	Graves' disease
30	22.0	18.5	19.0	15.0	16.0	12.5	14.0	11.5	3.0	3.5	6.0	6.0	8.0	7.0	†Meningioma—posterior and inferior orbit
31	14.5	8.5	10.0	4.5	8.5	2.0	7.0	1.0	4.5	4.0	6.0	6.5	7.5	7.5	†Mucocele—right frontal sinus
32	21.0	17.0	16.5	13.0	14.5	10.5	12.5	9.5	4.5	4.0	6.5	6.5	8.5	7.5	Undetermined
33	19.0	16.0	14.0	12.0	12.0	10.0	10.0	8.0	5.0	4.0	7.0	6.0	9.0	8.0	Undetermined
34	17.0	14.0	13.0	10.0	10.5	7.5	8.5	6.0	4.0	4.0	6.5	6.5	8.5	8.0	Graves' disease
35	23.5	16.5	19.0	11.5	16.0	8.5	14.5	7.0	4.5	5.0	7.5	8.0	9.0	9.5	Graves' disease
36	12.0	12.0	8.0	7.0	6.5	5.5	6.0	4.5	4.0	5.0	5.5	6.5	6.0	7.5	†Hemangioma—superior and anterior orbit

* Ex. = exophthalmic eye; N. = normal eye.
† Surgical and pathologic diagnosis was made in these cases; operation was not done in the others.

TABLE 5

MEAN POSTERIOR ORBITAL DISPLACEMENT (Y VALUES) DURING ORBITONOMETRY IN UNILATERAL EXOPHTHALMOS: 36 CASES

Pressure Factor	Mean Posterior Displacement (mm.)			
	Group 1	Group 2	Group 3	Normal
Y_{100}	1.9 (0.4)*	2.2 (0.3)	3.6 (0.8)	3.7 (0.7)
Y_{200}	3.1 (0.3)	4.1 (0.1)	5.6 (0.9)	5.8 (1.0)
Y_{300}	4.2 (0.4)	5.2 (0.2)	7.0 (1.1)	6.8 (1.2)

* Numbers in parentheses are standard deviations.

than for group 3, which has a slope almost identical with the normal. The same data are shown graphically in Figure 7.

The question was raised as to which, if any, of the pressures used is most significant. To decide this question, the increment of retrodisplacement in millimeters for each pressure used was determined, that is, Y_{100} minus Y_0, Y_{200} minus Y_{100}, and Y_{300} minus Y_{200} (table 6 and fig. 7). The values for groups 1 and 2 again varied from those of group 3 and the normal. Exertion of the first 100 gm. of pressure revealed the most significant difference in the groups, whereas the second 100 gm. showed less difference, and the third 100 gm. disclosed no appreciable difference between any of the groups and normal persons.

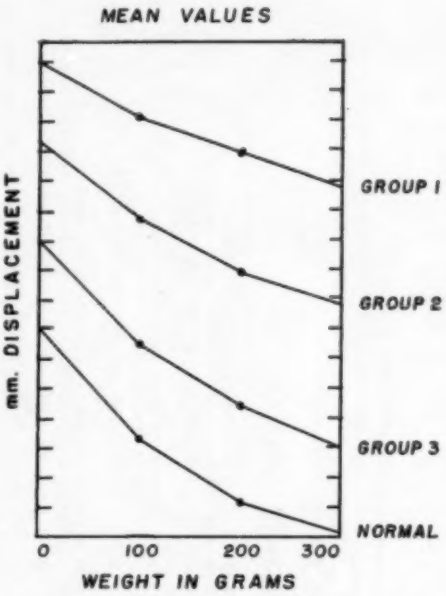


Fig. 6 (Dyer and Henderson). Curves for the mean Y values in each group and in normal eyes, showing variation in the degree of retrodisplacement.

COMMENT

Most of the new growths were located in the middle and posterior thirds of the orbit, the majority being in zones 2 and 3. Tumors in these locations, regardless of their type, caused increased resistance to retrodisplacement. Therefore, whether the lesion

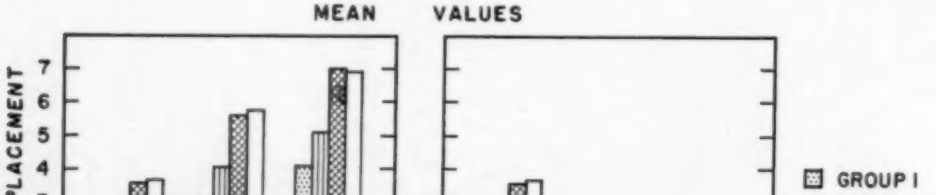


Fig. 7 (Dyer and Henderson). The left half represents mean Y values. The right half shows the mean retrodisplacement for each increment of pressure utilized.

TABLE 6

MEAN INCREMENT IN POSTERIOR ORBITAL DISPLACEMENT WITH EACH PRESSURE DURING ORBITONOMETRY IN UNILATERAL EXOPHTHALMOS: 36 CASES

Pressure Factor	Mean Increment in Posterior Displacement (mm.)			
	Group 1	Group 2	Group 3	Normal
Y_{100-0}	1.9 (0.4)*	2.2 (0.3)	3.6 (0.8)	3.7 (0.7)
$Y_{200-100}$	1.3 (1.3)	1.8 (1.9)	2.0 (2.1)	2.1 (2.1)
$Y_{300-200}$	1.0 (1.0)	1.1 (1.1)	1.4 (1.4)	1.0 (1.1)

* Numbers in parentheses are standard deviations.

was located temporally, nasally, inferiorly, or superiorly, posterior displacement was decreased in the presence of either a solid or a vascular tumor. This indicates that a posterior position inside the orbit is of more significance in reducing retrodisplacement than is the type of lesion. Lesions in such a position so filled the orbit that immediate resistance was encountered, with flattening of the first part of the curve. Copper considered that flattening in the middle and last portions of the curve was indicative of such lesions, but an early decrease in slope was the rule with definite orbital involvement in this series.

Patients who had tumors situated anteriorly in the orbit (division 1) in any of the zones or who had early Graves' disease yielded curves that were similar to the normal, indicating no interference with posterior displacement of the globe. Group 3 included a few cases in which tumors were located posteriorly in the orbit; in such instances, the growth apparently was not of such size as to interfere with posterior displacement of the globe. Kearns and associates indicated how advanced exophthalmos in Graves' disease might be associated with increased resistance, such as noted in group 2. They noted that the average orbital "tension" increased and the curve became flatter as the average degree of exophthalmos increased.

From these factors, it appears that a definitely increased orbital "tension" indicates the strong probability of a lesion in the posterior portion of the orbit, the location as to zone being uncertain. Patients with Graves' disease included in this category ordinarily would not present a problem, since other clinical signs would be present to aid in diagnosis. If orbitonometry reveals that the orbital "tension" is normal, little justification is lent to a diagnosis of an orbital new growth. In either case, a differential diagnosis of the type of tumor by this method is not possible, and the variety of lesions that can produce proptosis certainly behooves one to pursue diagnosis by other clinical means.

The most significant difference in orbital tension occurred during application of the first 100 gm. of pressure. Therefore, this is the most significant portion of the orbitonometric curve; the second phase is less important; and the use of 300 gm. of pressure yielded little or no information. The total compressibility of the globe did not yield diagnostic information of any great significance.

Our findings were essentially in accord with most of Copper's conclusions regarding orbital inflammation and new growths. Posteriorly situated tumors and diffuse edema or infiltration of tissue inhibited retrodisplacement to a maximal degree, the position of the mass being more important than whether it was firm or soft. Anterior tumors and mild edema produced the least limitation of displacement. In the absence of proptosis, it is less likely that one would utilize the orbitonometer; however, if the presence of a space-occupying lesion is suspected and increased orbital "tension" is noted, the likelihood of such a lesion is enhanced. The presence of proptosis and increased orbital resistance is evidence of an orbital space-occupying lesion, and repeated measurements showing either an increased resistance or no change would indicate either progression or a stationary state. Overlapping of findings will occur in some instances. It is these overlapping cases, such as in group 2, with its intermediate resistance to retrodisplacement,

and in cases in group 3 in which tumor is present and yet retrodisplacement is normal, that the orbitonometer fails to give a definitive answer in diagnosis.

Summary and Conclusions

A study has been made at the Mayo Clinic of the value of orbitonometry in 36 cases of unilateral exophthalmos arising from a variety of causes.

The orbitonometer did not prove to be of great value in the differential diagnosis of unilateral exophthalmos. A flattened curve of retrodisplacement of the globe is indicative of increased orbital "tension" and, therefore, is suggestive of orbital new growth, inflammation, or diffuse dense infiltration of tissue involving the posterior or middle divisions of the orbit or both, but it does not further define the location. The presence of a normal curve of displacement for the exophthalmic eye does not rule out the possibility of a space-occupying lesion within the orbit, since one may be situated anteriorly or may be too small to produce increased orbital "tension."

The presence of a group of cases in which displacement is intermediate indicates that orbitonometry will give equivocal results in some instances. Repeated measurements may be most useful in this group, since progression of exophthalmos as well as increasing resistance to retrodisplacement eventually may place the case in a more definitive category. Treatment in some form probably would be instituted before this stage was reached.

Flattening of the curve of ocular displacement, indicating increased orbital resistance, in its first phase (with the first 100 gm. of pressure) appears to be of the most significance, since the lesion has reached sufficient size to produce increased resistance at once. The use of pressure of more than 300 gm. apparently is not indicated.

The orbitonometer may be a useful adjunct to the diagnosis of orbital space-occupying lesions, but it cannot be relied on alone for diagnosis.

The findings in this series did not influence the decision of the physician in any case, since other clinical studies yielded satisfactory diagnostic results. It appears, therefore, that one would have a further bit of evidence in diagnosis if orbitonometry was utilized in unilateral exophthalmos, but its value as a routine office procedure is limited, and the evidence thus found would not be the determining factor with regard to surgical intervention. Clinical signs and symptoms and results of other routine studies continue to be the most reliable index in the diagnosis of unilateral exophthalmos.

Mayo Clinic.

References

1. Copper, A. C.: An Introduction to Clinical Orbitonometry. Leiden, H. E. Stenfert Kroese's Utigeners-MIJ. N.V., 1948.

2. Graefe, A. V.: Zusätze über intraoculare Tumoren. Arch. f. Ophth., **14**:103-144 (Abt. II; pt. I), 1868.

3. Langenhan: Instrumentelle Messung der Zurückdrängbarkeit des Augapfels in die Augenhöhle. Ztschr. f. Augenh., **24**:417-423, 1910.

4. Gutmann, A.: Piezometer, zur Diagnose retrobulbärer Orbitageschwülste. Ztschr. f. Augenh., **63**: 136-142, 1927.

5. ———: Physiologische Untersuchungen über Lageveränderung des Bulbus in der Orbita. Ztschr. f. Augenh., **31**:109-123, 1914.

6. Plegge, H.: Eine neue Methode zur Messung der Zurückdrängbarkeit des Bulbus in der Orbita (Piezometrie). Klin. Monatsbl. f. Augenh., **87**:584-596, 1931.

7. Georg, F.: Ein neues Piezometer. Arch. f. Ophth., **144**:68-80 (Dec.) 1941.

8. Means, J. H., and Stanbury, J. B.: Clinical orbitonometry. Am. J. M. Sc., **220**:357-361 (Oct.) 1950.

9. Galindez Iglesias, F.: Orbitonometría. Arch. Soc. oftal. hispano-am., **14**:141-165 (Feb.) 1954.

10. Grossmann, E. E., and Burns, T. A.: Clinical use of orbitonometry. Am. J. Ophth., **37**:520-528 (Apr.) 1954.

11. Kearns, T. P., Henderson, J. W., and Haines, S. F.: Clinical orbitonometry in Graves' disease. Am. J. Ophth., **36**:45-55 (Jan.) 1953.
12. Faldi, S.: Further clinical trials with Copper's orbitonometer. Gior. ital. oftal., **6**:498-502 (Sept.-Oct.) 1953.
13. Benedict, W. L.: Diseases of the orbit: Jackson memorial lecture. Am. J. Ophth., **33**:1-10 (Jan.) 1950.
14. Kearns, T. P. and Wagener, H. P.: Ophthalmologic diagnosis of meningiomas of the sphenoidal ridge. Am. J. M. Sc., **226**:221-228 (Aug.) 1953.

METASTATIC CARCINOMA OF THE IRIS*

DAHAR CURY, M.D.
Huntington Park, California

The eye is an uncommon site for metastatic new growths. These growths are usually multiple and often bilateral. To date eight histologically verified cases confined to the iris have been reported.[1-8] Microscopic examination of the primary site has been reported in three cases.[4-6]

It is the purpose of this paper to report two additional cases of metastatic carcinoma of the iris. In Case 1, the tumor, when first observed, clinically was confined to the iris but had extended to the adjacent ciliary body at the time of enucleation.

CASE REPORTS

CASE 1 (patient of Dr. Eric Richardson)

This 57-year-old white man complained of a dry, hacking cough for some months which began in the spring of 1952. A diagnosis of carcinoma of the right lung was made in June, 1952. On July 11, 1952, a right pneumonectomy was performed at St. Clare's Hospital. At the time of operation no metastases were found in the regional lymph nodes.

Two weeks after the operation and while the patient was still in the hospital the left eye became inflamed and painful. A small, grayish-white mass with many newly formed blood vessels was present on the temporal surface of the iris near the periphery. This mass grew in size and vascularity. The intraocular pressure was increased to 60 mm. Hg (Schiøtz). The eye was removed on September 18, 1952, under local anesthesia because of intractable pain.

The pathologic study of the resected lung revealed bronchogenic carcinoma.

The patient died in October, 1952, four months after the diagnosis of cancer of the lung had been made. There had occurred very rapid and widespread metastases, many of them to the bones of the skull and legs.

Pathologic report. Gross. A globe of normal size and shape is submitted. Sections are eccentric and only portions of the lens and anterior chamber are visible. That portion of the anterior chamber seen is filled with a dense heavy-staining mass which has indented the lens behind.

Microscopic. That portion of the cornea near the limbus is mainly seen in the sections studied. There is some vascularization in the middle and outer third. The endothelium is obscured for the most part by the mass in the anterior chamber.

The epithelium is intact. The stroma is not remarkable, and the angle meshwork is normal nasally. On the temporal side, the meshwork is compressed by the mass in the temporal angle. Some of the cells from this mass are seen in the Canal of Schlemm on this side.

Almost the entire chamber is filled with a tumor mass which appears to have emanated from the temporal iris and anterior portion of the ciliary body. The nasal angle is the only area not filled by the mass. This region contains an albuminous coagulum in which are floating a few tumor cells and red blood cells. The temporal iris is almost completely missing, having been destroyed by the tumor. The tumor has invaded the posterior chamber, temporally, and has exerted pressure against the lens. There is extensive necrosis throughout the tumor obscuring much of the cytology.

The tumor is composed chiefly of large, uniformly faint-staining cells with prominent small nucleoli. There are numerous mitotic figures. There is a suggestion of granular formation. Several large blood spaces are seen lined by tumor cells.

The tumor has caused a pressure necrosis in the medial half of the nasal iris. The iris has lost its normal crypts and folds. The stroma is marked by sanguineous extravasation and perivascular

* From the ENO Laboratory, New York Eye and Ear Infirmary, New York.

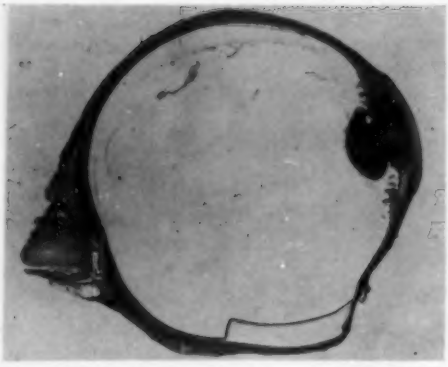


Fig. 1 (Cury). *Case 1.* This section shows the metastatic bronchogenic carcinoma from the lung filling the anterior chamber and infiltrating the iris.

round cell infiltration. The posterior pigment layer has lost many of its festoonations and is fragmented in places, especially medially, where the iris is connected by adhesive bands to the lens behind and to remnants of the temporal iris. The temporal iris, as noted, has been destroyed by hemorrhagic necrosis due to tumor invasion.

On the temporal side of the ciliary body, the tumor has invaded the anterior portion of the pars corona. There is necrosis in these processes. The ciliary epithelium and pigment epithelium have been destroyed or are barely distinguishable. There is vascular congestion in the ciliary body.

There is vascular congestion in the larger vessels of the choroid. The lamina vitrea and pigment epithelium are intact.

There is some gliosis in the nerve fiber and ganglion cell layers. Marked cystic degeneration is seen at the temporal ora serrata. The optic nerve and the papillae are not seen in the sections examined.

The vitreous is detached posteriorly and in places is filled with a fibrinous coagulum. The lens is markedly indented in the temporal anterior quadrant. The capsule is intact, but there is pressure necrosis in the subcapsular epithelium, underlying cortex, and nucleus, and in these areas there are vacuoles.

Impression. Metastatic bronchogenic carcinoma (epidermoid?) into the iris and ciliary body. Necrosis of iris and ciliary body. Iridocyclitis. Complicated cataract.

CASE 2 (patient of Dr. Leon Weiss)

This 79-year-old white man had a blind and painful right eye when first seen at the New York Eye and Ear Infirmary in June, 1954, at which time he was admitted for an enucleation. The clinical diagnosis was amaurosis, iridocyclitis, secondary glaucoma, retinal detachment, and cataract. The iridocyclitis with secondary glaucoma had been uncontrolled by medication for three weeks prior to admission. The fellow eye showed retinal degen-

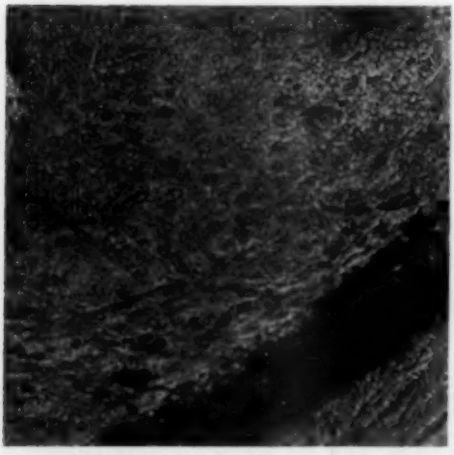


Fig. 2 (Cury). *Case 1.* High-power view of Figure 1, showing morphology of the metastatic bronchogenic carcinoma from the lung.

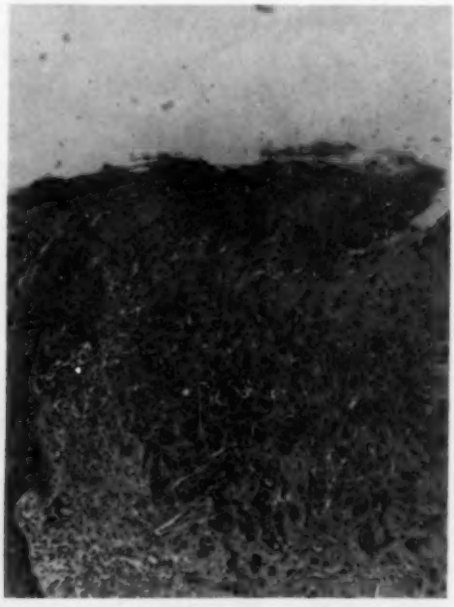


Fig. 3 (Cury). *Case 2.* Low-power view of original lesion of squamous-cell carcinoma of the lower lip.

erative changes, lenticular opacities, and vitreous floaters.

Seven years earlier (June, 1947) squamous cell carcinoma of the lower lip was diagnosed and excised at Memorial Hospital, New York City. Periodic examinations revealed no recurrence. His last examination was in April, 1954, two months before his admission to the New York Eye and Ear Infirmary, at which time there was no systemic evidence of recurrence.

Pathologic study. Gross. An eye measuring 25 by 24 mm. is presented for study. The eye is somewhat irregular in shape and does not show an external scleral sulcus as it is usually seen. The sections are cut somewhat eccentrically and do not show the pupillary area or pass directly through the nerve. The anterior chamber contains a reddish coagulum. The lens is oblong in shape, its center removed during fixation.

Microscopic. The corneal epithelium is intact. It contains vesicles in its outer layer and considerable edema of the basal layers. Bowman's membrane and Descemet's membrane are identified. There are some bullae between Bowman's membrane and the overlying epithelium. The stroma is edematous. The endothelium is edematous and is continuous with an epitheliumlike membrane on the surface of the iris at the pseudo angle. A granular coagulum is adherent to its inner surface.

There are peripheral anterior synechias of the iris. The iris does not show crpyts and folds. Festooning of the pigment epithelium is present but irregular. There are small posterior synechias between the pigment epithelium of the iris and the lens. The iris shows atrophy and mobilization of its pigment. There is a membrane on much of the anterior surface of the iris, especially adjacent to the pseudo angle. The cells of this membrane have the appearance of epithelial cells and are arranged

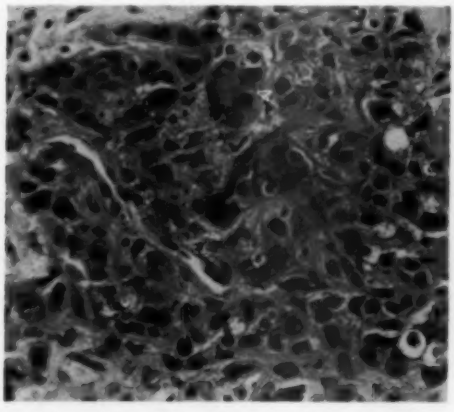


Fig. 4 (Cury). *Case 2.* High-power view of section from the lip.

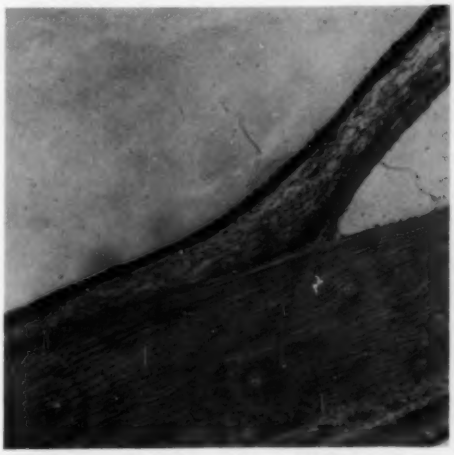


Fig. 5 (Cury). *Case 2.* Metastatic squamous carcinoma from the lower lip on the surface of the iris and extending to the pseudo angle.

in irregular layers, and in some areas penetrate the stroma of the iris. The nuclei are oval-shaped and contain numerous nucleoli. Some of the nuclei are hyperchromatic. The cytoplasm is scant in many of the cells studied. Occasional mitotic figures are seen. The ciliary body shows mobilization of its pigment epithelium, and at the ora serrata there is a "ringschwiele."

The choroid is compressed, and in portions is

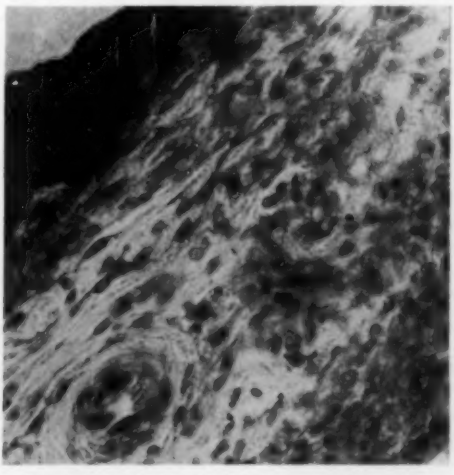


Fig. 6 (Cury). *Case 2.* High-power view of Figure 5, showing metastatic squamous cell carcinoma infiltrating the stroma of the iris.

infiltrated with chronic inflammatory cells. The pigment epithelium is adherent to the choroid. Some of the pigment epithelium cells have migrated into the subretinal fluid. There are numerous pathologic drusen.

The retina is detached except at the optic disc and the ora serrata and shows degeneration and atrophy. Numerous macrophages containing pigment are seen in the retina. Some foci and diffuse infiltration of chronic inflammatory cells are seen. The retina is not seen in its entirety, probably artefact of fixation, but that portion seen is separated and thrown into folds. There is cystoid degeneration of the retina. The retinal elements have undergone degeneration and atrophy. The subretinal fluid is a clear eosinophilic staining coagulum which contains pigmented macrophages and a few cholesterol clefts. The optic nerve shows complete irregularity of its nuclear columns. There is an increase in the glial elements.

The vitreous body lies anteriorly. It contains more macrophages than are usually seen and shows areas of fresh hemorrhage. Behind the lens there are numerous areas which show a coarse coagulum thought to be lens substance that has apparently leaked from the hypermature lens. Numerous macrophages and an occasional eosinophil are seen in these areas.

The crystalline lens is shrunken and there is a break in the continuity of the lens capsule posteriorly. The lens substance shows liquefaction and in some areas stains basophilically and contains crystals. At the equator of the lens, the lens capsule is drawn upward toward the ciliary body and surrounds several inclusions of lens substance. On the posterior surface of the lens there are collections of pigment and macrophages containing pigment.

The anterior chamber contains a granular eosinophilic coagulum. The angle is obliterated and a pseudo angle is seen. Schlemm's canal is not identified in the sections studied.

Impression. Metastatic squamous-cell carcinoma of the iris. Separation of the retina. Secondary glaucoma. Chronic uveitis. Optic atrophy due to glaucoma. Hypermature lens with lens reaction.

Summary

Two additional cases of metastatic carcinoma of the iris are reported in which the enucleated eye and primary site were examined histologically. The second case is remarkable in that it resembles epithelization of the anterior surface of the iris as seen in downgrowths in the anterior chamber.

2665 Walnut Street.

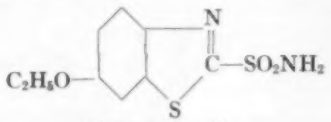
References

1. Bollack, J., Bertillon, F., and Roques, A.: Metastatic epithelioma of the iris. Ann. Ocul., **164**:336, 1927.
2. Hudson, A. C., and Lister, W.: Metastatic hypernephroma of the iris. Proc. Roy. Soc. Med., **27**:1613, 1934.
3. Sanders, T. E.: Metastatic carcinoma of the iris. Am. J. Ophth., **21**:646, 1938.
4. Asbury, M. K., and Vail, D.: Metastatic carcinoma of the iris. Am. J. Ophth., **23**:402, 1940.
5. Sautter, H.: Some interesting findings on the iris. Klin. Monatsbl. f. Augenh., **113**:44, 1948.
6. Reese, A. B.: Tumors of the Eye. New York, Hoeber, 1950.
7. Greear, J.: Metastatic carcinoma of the eye. Am. J. Ophth., **33**:1015, 1950.
8. Greer, C. H.: Metastatic carcinoma of the iris. Brit. J. Ophth., **38**:699, 1954.

USE OF A NEW CARBONIC ANHYDRASE INHIBITOR (CARDRASE) IN GLAUCOMA

ADOLPH POSNER, M.D.
New York

Cardrase* is a potent carbonic anhydrase inhibitor which has been demonstrated to be an effective diuretic when administered orally.[1] Its pharmacologic action is similar to that of acetazolamide. In animal and clinical studies, the diuretic action of Cardrase has been shown to be twice as potent on a milligram basis as acetazolamide.[2] Chemically it is 6-ethoxybenzothiazole-2-sulfonamide, and has the following structural formula.[3]



Ethoxzolamide

This report deals with the action of Cardrase on the ocular tension in various types of glaucoma. The drug was administered to 35 patients but the case material for this study consists of 20 patients from my private practice. The remainder could not be followed for a sufficient period. All these patients previously had been thoroughly investigated to determine the type of glaucoma and its response to the use of miotics. In eight cases, Diamox had been used previously so that an approximate comparison between the two drugs, as to effectiveness, could be made. Several patients had been carefully followed over long periods of time —in some cases as long as 20 years. All tension measurements were performed by me using the same certified Schiøtz-Sklar tonometer. The tensions were measured at the same time of day and, in each case, the time of the last instillation of miotic and the last dose of Cardrase was noted on the patient's record.

Table 1 gives a simplified summary of the results of this study. The case material consists of 12 cases of chronic simple (open-angle) glaucoma, three cases of chronic non-congestive angle-closure glaucoma, one case of secondary glaucoma caused by peripheral anterior synechias secondary to surgery for angle-closure glaucoma, one case of secondary glaucoma caused by essential atrophy of the iris, one case of secondary glaucoma caused by posterior dislocation of the lens (Marfan's syndrome), one case of pigmentary glaucoma, and one case of congenital glaucoma in a microphthalmic eye complicated by cataract surgery.

Cardrase was administered in minimal doses and in most cases was found to be effective in lowering the tension to the desired level. For purposes of comparison, Cardrase was given in doses one-half those of Diamox to the patients who had previously used Diamox. Only in two patients (T. B. and M. B.) was it necessary to increase the dosage.

In the first case, one tablet (125 mg.) of Cardrase twice a day was required to maintain the tension at the level attained previously by Diamox, one-half tablet (125 mg). twice a day.

In the second case, Cardrase (62.5 mg.) had the same immediate effect as Diamox (125 mg.) but its effect lasted only four hours, whereas the Diamox effect had lasted eight hours. This patient became subjectively aware of moderate elevations of tension by the onset of blurring of vision which would occur whenever the tension exceeded 30 mm. Hg.

In the other instances (five cases) in which the effect of Cardrase could be compared

* Cardrase was supplied as ethoxzolamide in tablets of 125 mg. through the courtesy of Dr. E. W. Young, Medical Division, Department of Clinical Investigation, The Upjohn Company, Kalamazoo, Michigan.

TABLE 1

SUMMARY OF RESULTS WITH CARDRASE

Name	Diagnosis	Dosage	Tolerance	Effectiveness in Control of Tension	Comparison with Diamox (equal dosage)
A. B.	Chronic simple glaucoma	62.5 mg. b.i.d.	Good	+	2×
T. B.	Chronic simple glaucoma	125 mg. b.i.d.	Good	+	1×
M. M.	Chronic simple glaucoma	31 mg. q.4.h.	Good	+	—
T. S.	Chronic simple glaucoma	62.5 mg. b.i.d.	Good	++	2× (or more)
H. J.	Chronic simple glaucoma	31 mg. b.i.d.	Poor	0	—
W. G.	Chronic simple glaucoma	62.5 mg. b.i.d.	Good	+	2×
A. L.	Chronic simple glaucoma	62.5 mg. b.i.d.	Good	0	—
L. L.	Chronic simple glaucoma	62.5 mg. b.i.d.	Good	+	2×
A. L.	Chronic simple glaucoma	62.5 mg. t.i.d.	Good	++	—
M. C.	Chronic simple glaucoma	62.5 mg. 4.i.d.	Good	+	—
S. M.	Chronic simple glaucoma	62.5 mg. 2.i.d.	Good	±	—
J. K.	Chronic simple glaucoma	62.5 mg. 4.i.d.	Good	+	2× (Surgery performed because of marked fluctuations of tension)
G. N.	Chronic angle-closure glaucoma	62.5 mg. b.i.d.	Good	++	—
T. B.	Chronic angle-closure glaucoma	62.5 mg. b.i.d.	Good	++	—
C. L.	Chronic angle-closure glaucoma	62.5 mg. 4.i.d.	Good	++	—
G. K.	Secondary glaucoma (caused by peripheral anterior synechias)	62.5 mg. 4.i.d.	Good	0	—
C. K.	Secondary glaucoma (essen. atrophy of iris)	62.5 mg. t.i.d.	Poor	0	—
F. W.	Secondary glaucoma (dislocated lenses)	31 mg. b.i.d.	Poor	+	1× (Diamox also was poorly tolerated)
B. L.	Pigmentary glaucoma	62.5 mg. t.i.d.	Good	++	—
M. B.	Congenital microphthalmia and glaucoma	62.5 mg. t.i.d.	Good	+	1×

Not effective 0
Moderately +
Very effective ++
Dash indicates data for comparison not available.

with that of Diamox, one tablet (125 mg.) of Cardrase appeared to be equivalent to one tablet (250 mg.) of Diamox. Cardrase was generally well tolerated. Only two patients had to discontinue its use because of side effects, especially nausea and weakness. Both patients previously had been unable to tolerate even minute doses of Diamox (62.5 mg.). The side-effects produced by both drugs were similar.

The present study corroborates the previously reported findings that Cardrase is effective as a carbonic anhydrase inhibitor in doses approximately one-half those of Diamox, and has a tension-lowering action in glaucomatous eyes regardless of the nature or cause of the glaucoma. This action is similar to that of Diamox and in most cases appears to be twice as great. Symptoms of intolerance to the drug were noted in two out of 20 cases and these were similar to those experienced previously by the same patients when using Diamox. The symptoms disappeared immediately upon discontinuance of the drug and left no after-effects. The majority of the patients in this study have been using Cardrase for periods of six to 12 months. No untoward effects attributable to the drug were noted in any of the patients. Mild paresthesias of the fingers and toes occurred in some patients at the beginning of treatment but disappeared later.

SUMMARY

1. Cardrase (6-ethoxybenzothiazole-2-sulfonamide), a new carbonic anhydrase inhibitor, is effective in lowering ocular tension in glaucoma.

2. Cardrase may be administered orally in dosages of 62.5 to 125 mg. two to four times daily. It is generally well tolerated and has shown no deleterious side effects.

3. In 20 cases of glaucoma, including chronic simple, angle-closure, and secondary glaucomas, Cardrase proved to be twice as potent and equally well tolerated as Diamox.

667 Madison Avenue (21).

REFERENCES

1. Ford, R. V., Spurr, C. L., and Moyer, J. H.: The problem of bioassay and comparative potency of diuretics: II. Carbonic anhydrase inhibitors as oral diuretics. Circulation, **16**:394, 1957.
2. Korman, J.: Carbonic anhydrase inhibitors: I. Benzothiazole derivatives. In press.
3. Ibid.

RECENT ADVANCES IN THE MANAGEMENT OF RETINAL DETACHMENT*

GUILLERMO PICÓ, M.D.
Santurce, Puerto Rico

The modern management of retinal detachment can fairly be said to date from 1929. In that year Gonin[1] established the importance of the retinal break in the pathogenesis of this process and also pointed out that successful therapy must be based upon closure of this opening. He had made the same points in an earlier paper, published in 1920, which had attracted little attention.

A variety of methods have been devised to accomplish closure of the retinal tear. They include cauterization with the actual cautery, which Gonin employed; chemical cauterization by means of scleral trephinations; diathermy; cathode electrolysis; and various surgical techniques. All of these methods have the same purposes, to close the tear in the retina by inducing a regional chorioretinitis and to provide drainage for the subretinal fluid that prevents reattachment of the retina. Some supplementary procedures have also been devised, such as the injection of air or physiologic salt solution into the vitreous, in order to push the retina toward the treated choroid.

All of the earlier methods devised to close the tear in the retina had some successes but there were a great many failures, all for the same reason, that the presence of vitreoretinal adhesions prevented the retina from settling down and becoming reattached to the treated choroid. This has been repeatedly demonstrated by studies with the Hruby lens and the slitlamp, as well as by histologic studies on enucleated eyes from both human subjects and experimental animals.

With the Hruby lens and the slitlamp it is often possible to visualize the adhesions and other vitreous changes responsible for the retinal break and of major importance in both the prognosis and the decision concerning the operation to be selected in each case.

In 1950, Paton[2] commented on how little improvement had occurred in the surgical management of retinal detachment over the preceding several years. Since 1950, few fields of ocular surgery have witnessed more important advances.

PREOPERATIVE STUDIES

One reason for recent improvement in the results of surgery for retinal detachments has been more accurate localization of the tear before operation. Ophthalmoscopic localization, with charting of all breaks, fixed retinal folds, and other details, is now recognized as essential for the success of surgery.

In the past, although indirect ophthalmoscopy was widely used elsewhere, most sur-

* From the Department of Ophthalmology, University of Puerto Rico School of Medicine. Presented at the sectional meeting of the American College of Surgeons, New Orleans, Louisiana, February, 1957.

geons in the United States confined their preoperative studies to the direct technique. In 1949, Arruga[3] pointed out the importance of employing both techniques in all cases. In general, he stated, the direct method is likely to be more useful in differentiating hemorrhages from retinal breaks and in comparing differences in retinal elevation. The indirect method, on the other hand, permits examination of a larger field, and only by this technique can the periphery of the retina be examined. Stronger illumination is also possible with the indirect method.

Schepens[4] also insists on the need for the indirect method in examination of the ora serrata, where, in his opinion, most breaks are located. The indirect stereoscopic ophthalmoscope which he devised has increased interest in the indirect technique, as has his revival of Trantas' technique of examining the periphery of the fundus. By this technique, pressure is applied to the sclera in the region of the ora serrata, either on the eyelids or directly. The scleral depressor which Schepens devised for this purpose (a small curved rod mounted on a thimble) is used in combination with his indirect ophthalmoscope.

Surgical techniques

Over the past several years, large series of cases have been published showing good results with the classical diathermy operation in 75 percent or more of all cases of retinal detachment. The desire for better results, however, particularly in cases in which the prognosis is poor, has led to the introduction of a number of new surgical techniques based on the principle of shortening the sclera.

Penetrating (full-thickness) scleral resection

A so-called penetrating type of operation designed to shorten the sclera was described by Müller in 1903, but it had only a limited acceptance. The principle upon which it was based was sound, but because it was frequently associated with loss of vitreous and vitreous hemorrhages it was reserved for complicated cases, in which the outlook was practically hopeless. The operation was revived and revised by Lindner in 1933,[5] and good results have recently been reported with it by Pischel,[6] Vail,[7] Borley,[8] and Kronfeld and Pischel[9] in this country.

In 1949, Weve[10] described another procedure for shortening the sclera, consisting of full-thickness reefing or infolding achieved by a running catgut suture placed intrasclerally. The operation is concluded by the application of diathermy over the retinal break.

Lamellar scleral resection

Lindner,[11] in 1946, described the operation of lamellar scleral resection, which he had devised as a matter of expediency: In a case in which he had planned to do a full-thickness resection, the sclera was so thin that the choroid was perforated with the initial dissection of the strip of sclera to be excised. He therefore removed the superficial layers and folded the scleral strip inward when the sutures were tied. In the six cases in which he used this technique, Lindner found that it simplified the shortening operation. Dellaporta,[12] in 1956, reported that Lindner had abandoned this operation because he found, in some cases which required reoperation, that the sclera had unfolded. Dellaporta[13] himself, in 1951, had reported encouraging results with this method, and good results with lamellar scleral resection were also reported by Shapland,[14] Paufique and Hugonnier,[15] and Berliner.[16]

Lamellar scleral resection has undergone many modifications since it was first introduced, but the fundamental steps of the procedure, which is preferably performed under general anesthesia, are as follows:

A large conjunctival flap is dissected, with its base at the limbus. Good exposure is thus provided for the area of sclera to be

resected. One or more recti muscles are cut free from the sclera, the number depending upon the extent of the exposure necessary. Sutures of 4-0 chromic catgut are applied before the muscle is cut.

The retinal breaks are localized by ophthalmoscopy. While the surgeon focuses the light on the tear, his assistant applies gentle pressure to the sclera with a hook. The first application is made at the site at which the light shines through the sclera and the hook is moved until the depression which corresponds with the area of the tear is clearly evident to the surgeon. This area is then marked out by gentian violet applied on a cotton applicator or by the light application of a diathermy electrode.

The extent of the strip to be resected is next marked off by parallel marks with the knife. Usually the area is three to four-mm. wide. Whether it is limited to a single quadrant or extends over all four quadrants depends upon the number, size, and location of the retinal tears and the presence of other pathologic processes. As a rule, the resection is parallel to the equator and involves half of the circumference, though it may be necessary to extend the resection to include the entire circumference of the eyeball.

The midpoint of the strip to be resected should correspond to the location of the retinal breaks or lie slightly posterior to them. In any event, all the breaks should be anterior to the resected sclera.

The parallel horizontal lines which indicate the area to be resected are connected in the middle by an anteroposterior incision. Dissection is begun at this point. The outer four-fifths of the scleral lamella is freed with the knife, all that remains being a thin layer of light grayish tissue which practically corresponds to the lamina fusca. The edges are undermined as dissection is completed toward each side. Care must be taken not to carry the dissection too deep and risk perforating the choroid.

Mattress sutures of 4-0 black braided silk are inserted through the lips of the resected area. Grieshaber needles are used, or threaded Davis and Geck needles, which are equally satisfactory.

Light surface diathermy is then applied with a small ball or blunt electrode all along the area of lamellar scleral resection. Excessive electrocoagulation should be avoided; this can be determined by the dark gray discoloration produced when the electrode is applied to the scleral lamella left in situ. Some surgeons apply additional diathermy anterior or posterior to the area of resection, but this is not necessary if the retinal breaks have been well localized and the resected area includes the tears or is posterior to them.

Surface coagulation is next applied from the tips of the resected area of sclera to the ora serrata, in order to prevent extension of the retinal detachment if seepage of subretinal fluid should occur through any breaks which might remain open anterior to the area of scleral resection.

Drainage of subretinal fluid is accomplished by a small puncture with a needle or an electrode in the base of the resected area. To obtain complete evacuation of the fluid it may be necessary to enlarge the puncture with the tip of a lacrimal dilator.

The mattress sutures are tied, thus closing the area in which the resection was performed and infolding the scleral lamella and the choroid left in this area. It may be extremely difficult, when an extensive resection has been performed, to tie these if hypotony is not present. In such cases it may be necessary to perform repeated paracenteses of the anterior chamber, a procedure that is, however, not always harmless and that should be avoided when possible. The rectus muscles which were cut are reattached to their respective stumps or are recessed one or two mm., as may be necessary, to avoid postoperative muscular imbalance. Finally, the conjunctiva is closed with a continuous suture of 6-0 silk.

Scleral buckling

Schepens contributed several of the details of the technique of lamellar resection just described. In 1955, at a symposium on retinal detachment in New York,[17] he described another modification of the operation, the implantation of a polyethylene tube 1.5 mm. in diameter in the area of resection. The tube is invaginated with the choroid and the scleral lamella left in situ when the sutures are tied, a more pronounced buckling thus being produced than when the operation is done without the tube. It is important in this technique that the midpoint of the area of excision be at the site of the retinal break (breaks) or just posterior to it. The operation is concluded, as in lamellar resection, by the application of surface diathermy and the release of subretinal fluid.

In 1956, Chamlin and Rubner[18] described another technique of scleral buckling under the name of lamellar undermining. The dissected strip of scleral lamella is left attached to the base of the scleral groove along a one-mm. area in the center and is infolded when the sutures are tied. These observers found experimentally that the scar thus produced is firmer than the scar produced in the tube technique, apparently because the raw scleral surfaces tend to encourage scar formation and discourage the tendency of the tissues to unfold.

Scleral outfolding

Everett's[19] technique for scleral shortening, which was described in 1955, was not original with him, as he thought; a similar operation had been developed independently by Castroviejo.[20] The procedure consists of the formation of an external fold of sclera by the application of mattress sutures, the extent and location of the fold being determined by the state of the retina and vitreous. The globe is decompressed, several times if necessary, by release of the subretinal fluid through penetrating diathermy punctures. The sclera is then raised by means of two forceps of the Lester type, and mattress sutures are introduced to create a fold of the desired length. Diathermy is applied around the fold, to produce a chorioretinitis. The folded sclera distal to the sutures is excised with scissors after the fold has been completed.

In his original technique, Everett separated the choroid from the sclera by introducing a spatula through a small scleral incision before introducing the sutures. Later,[21] he eliminated this step. In experimental studies on rabbits, he had observed that choroidal hemorrhage was more frequent in this operation than in lamellar resection and the polyethylene-tube technique, and he concluded that it was the separation of the sclera and choroid by the spatula which was responsible for the bleeding. His experimental studies showed that the introduction of the spatula was not really necessary, since it was possible to determine without it that the choroid had been separated from the scleral fold.

In 1956, Castroviejo[20] modified his operation of scleral outfolding by employing metal clips instead of sutures to hold the scleral fold. He emphasized that this technique was still in the testing stage. The results he had obtained with it in five cases, in most of which the prognosis was unfavorable, compared favorably with results obtained by other, more complicated, scleral shortening operations. There was, however, a persistent conjunctivitis, which did not respond to antibiotics or cortisone, in two cases, and he feared that it might have been induced by the use of the clips.

Vitreous implant

In 1956, Shafer[22] suggested the addition to the classical diathermy operation of another step, the injection of sterile human vitreous into the vitreous chamber. The injection is carried out by means of an 18-gauge needle through a sclerotomy at the pars plana; the incision is closed by mattress sutures after the injection is completed. The vitreous used could more correctly be described as a vitre-

ous aspirate. It is not the usual jellylike vitreous but a clear, transparent, oily fluid. Shafer believes that this vitreous is retained long enough to keep the amount of vitreous at the normal level (that is, the volume before detachment of the retina), while air and saline solution injected for this purpose are absorbed within a few days.

Shafer's observation that fixed retinal folds and vitreous bands frequently disappear after a vitreous implant may be of great significance. It suggests the possible presence in the injected vitreous of some factor which destroys the vitreoretinal bands presumably responsible for the detachment. Whatever the explanation, Shafer's results were good in a number of cases in which other techniques had failed.

Critique of surgical techniques

An analysis of the literature of scleral shortening operations indicates that lamellar scleral resection is the most widely favored procedure, whether it is performed with or without the added step of scleral buckling with a polyethylene tube. With the present emphasis upon vitreous traction as the cause of retinal detachment,[23] it seems logical to treat unfavorable cases by a procedure which will bring the treated choroid to the detached retina and thus nullify the traction effects of vitreoretinal adhesions. The objective of both procedures is the same, to form an internal fold composed of treated choroid and scleral lamella; the use of the polyethylene tube simply adds bulk to the fold.

Dellaporta[24] confirmed this observation in experimental studies on dogs. He also showed mathematically that the increase of volume produced in the interior of the eye by the buckling operation is considerably greater than the increase which occurs in lamellar resection. Everett's[21] experimental studies in rabbits are to the same effect, though he thought that a more long-lasting reduction in the scleral area was produced by his outfolding operation. The obvious disadvantage of his technique is the absence of the beneficial effects produced by inward protrusion of the sclerochoroidal wall toward the detached retina. This effect is present in both of the other types of scleral shortening which have been discussed.

For the present, and until reports of much larger series are available, the ophthalmic surgeon must make his own decision about the technique he employs. Possibly the smaller infolding of the sclerochoroidal wall secured in lamellar resection alone may be sufficient in the cases in which the retina is not pulled too far away from the choroid. The buckling operation, however, seems indispensable in cases in which the separation is more extensive. Probably the tendency for the fold to disappear in both operations is not a very important factor in the end results. By the time this has occurred, weeks or months after the fold has been formed, the chorioretinal scar should be so firm that redetachment will not occur.

Indications for scleral shortening

The classical diathermy operation is now usually reserved for cases in which the retina settles back into position after three or four days of rest in bed and in which examination with the ophthalmoscope and slitlamp shows no evidence of vitreous traction. While there may still be some disagreement concerning all the indications for scleral shortening, a certain number are well established:

1. *Inability of the retina to settle back* reasonably well after several days of immobilization in bed with both eyes covered. There are three possible causes, vitreous traction, the inability of the choroid to absorb the subretinal fluid, and the presence of numerous large retinal tears.

Schepens[23] has given an excellent description of the fundal changes in retinal detachment which provide evidence of traction by vitreoretinal adhesions. The first proof is the presence of fixed folds which do not undulate as the eye is moved and do not flatten out with bedrest. They are frequently observed as circular folds in the equatorial

region or as starshaped folds in any area of the fundus. The second proof of traction is the occurrence of massive vitreous retraction. When the whole retina is detached, fixed folds radiate from the disc. The third proof is the presence of tears with rolled-up, convex edges; these folds, like fixed folds, do not flatten out with bedrest. If the diathermy operation is done in the presence of any of these three findings, the retina will remain detached even if the subretinal fluid is drained or if air or saline solution is injected into the vitreous.

If there is a suspicion that failure of the patient to improve on strict bedrest is due to inability of the choroid to absorb the subretinal fluid, a penetrating puncture may be done at the beginning of the classical diathermy operation, for drainage purposes. If observation with the ophthalmoscope then shows a considerable diminution of the detachment, the diathermy operation may safely be proceeded with. Otherwise, a lamellar scleral resection is indicated, with or without introduction of a polyethylene tube. The surgeon must bear in mind, however, that the hypotony produced by early drainage of the subretinal fluid may make the other steps of the classical diathermy operation difficult, as well as the performance of scleral resection.

2. *Aphakia.* It is now well known that the prognosis in retinal detachment is worse in an aphakic patient, especially if vitreous is lost at operation. In these cases, vitreous bands are apparently responsible for the pull on the retina and its subsequent detachment; this is true even if there has been no vitreous loss. If the retina settles back with bedrest, the classical diathermy operation may be successful. If it does not, the presumption of vitreous retraction or traction is so strong that a scleral shortening operation is indicated, preferably by the buckling technique.

3. *High myopia.* The prognosis in these cases is usually poor because degenerative retinal and vitreous changes frequently accompany retinal detachment. A scleral staphyloma may also form. Better results are obtained in such cases by one of the scleral shortening operations than by diathermy.

4. *Multiple retinal breaks.* These are usually associated with degenerative atrophic changes in the retina and are better treated by the walling off accomplished by a shortening operation than by the usual multiple applications of diathermy to the tears.

5. *Large peripheral breaks or disinsertions.* These should always be treated by a scleral shortening operation.

6. *Retinal detachments following penetrating wounds,* intraocular foreign bodies, or vitreous hemorrhages. In these cases traction by vitreous bands is usually responsible for the detachment, and the chances of success are better with a shortening operation.

7. *The necessity for reoperation.* When a previous, competently performed diathermy operation has failed, it is reasonable to assume either that this type of operation was not indicated originally or that the diathermy application has produced changes in the vitreous and retina which require scleral shortening. The shortening operation, unfortunately, is more difficult as a secondary than a primary procedure because of the scleral and choroidal changes produced by the previous coagulation, and both perforation and loss of vitreous are distinct possibilities.

Preoperative and postoperative management

Most ophthalmologists continue to apply bandages to both eyes, or at least to prescribe pinhole glasses, and to immobilize the patient in bed for 48 hours or more before surgery for retinal detachment. Schepens[17] permits his patients to be ambulatory and does not occlude the eyes. He believes that flattening of the detachment before a buckling operation is undesirable because it may

diminish the quantity of subretinal fluid present and thus make the procedure both more difficult and less effective.

There are wider differences of opinion about postoperative management.[17] The great majority of ophthalmologists (of whom I am one) continue to be conservative. They keep both eyes bandaged and keep the patients recumbent in bed for at least 14 days; they then permit them to sit up in bed at intervals for the next week. When the patients become ambulatory, pinhole glasses are prescribed. Arruga[25] and Shipman[26] both regard this as the ideal routine.

Graham Clark,[17] on the other hand, keeps his patients in bed for only a day. The eyes are bandaged for six days, and pinhole glasses are worn for a month after operation. His theory is that the chief factor that promotes separation of the retina is the movements of the eye; the essential consideration, therefore, in the prevention of redetachment is not immobilization of the body but immobilization of the eyes only.

Schepens[17] considers that the use of an eye bandage and maintenance of bedrest are not necessary for more than four to eight days after a buckling operation, though more caution is necessary after the classical diathermy operation. When the patient who has undergone a buckling operation becomes ambulatory, he wears dark glasses but not pinhole glasses.

In my own opinion,[27] immobilization for 14 to 21 days after operation does not seem excessive if one recollects the effects of electrocoagulation on the choroid from the initial formation of the choroidal exudate to the initial formation of a firm chorioretinal scar. It may be that Clark is correct and that immobilization of the eyes, without immobilization of the head and neck, is sufficient to prevent redetachment of the retina in the postoperative period. Schepens' buckling operation facilitates the formation of chorioretinal adhesions and healing because it achieves a closer approximation of the choroid to the retina than is accomplished in the diathermy operation. Perhaps, therefore, the four to eight days of bedrest which he advises is really sufficient when his technique is used.

On the other hand, the principles of wound healing remain the same, regardless of the surgical technique employed, and it requires almost 14 days for a firm chorioretinal scar to form. It therefore seems entirely reasonable to assume that even better results might be obtained with the scleral buckling operation than are now being achieved if the usual conservative policy of postoperative immobilization were universally practiced. Only the comparative study of large series of cases will produce the final answer concerning the optimum period of immobilization of eyes and body after surgery for retinal detachment.

Summary

The modern management of retinal detachment began in 1929, with the demonstration that the retinal break was fundamental in the pathogenesis of the condition and that successful therapy must be based upon closure of the tear.

Preoperative ophthalmoscopic localization of the tears explains part of the recent improvement in surgical results. Both the direct and the indirect techniques should be employed.

Good results are still being obtained with the classical diathermy operation in 75 percent or more of all retinal detachments. A number of surgical techniques, all based on the principle of shortening the sclera, are now available for use in cases in which the prognosis is poor or in which, for other reasons, the diathermy operation is not likely to be successful.

The most popular of these new operations is lamellar scleral resection, which is often performed with an added step, scleral buckling produced by the use of a polyethylene tube. This procedure is logical because it

brings the treated choroid to the detached retina and thus nullifies the traction effects of vitreoretinal adhesions. The combined operation seems essential in extensive separations.

Although some ophthalmologic surgeons, for apparently good reasons, are shortening the period of bedrest after surgery for retinal detachment, conservatism still seems wisest, in view of the 14-day period necessary to achieve wound healing, regardless of the technique employed.

The indications for scleral shortening are presented, and a standard technique is described.

Avenida Ponce de Leon, 654.

References

1. Gonin, J.: Tr. Internat. Cong. Ophth., **2**:661, 1929.
2. Paton, R. T.: Recent advances in the surgery of retinal detachment. M. Rec. & Ann., **44**:66, 1950.
3. Arruga, H.: Relative importance of direct and indirect ophthalmoscopic examination in treatment of retinal detachment. Brit. J. Ophth., **33**:651, 1949.
4. Schepens, C. L.: Progress in detachment surgery. Tr. Am. Acad. Ophth., **55**:607, 1951.
5. Lindner, K.: Heilungsversuche bei prognostisch ungünstigen Fällen von Netzhautabhebung. Ztschr. f. Augenh., **81**:277, 1933.
6. Pischel, D. K.: Basic principles of retinal detachment operations, with special reference to eyeball shortening operation. Tr. Am. Acad. Ophth., **49**:155, 1945.
7. Vail, D.: Scleral-resection (eyeball-shortening) operation. Am. J. Ophth., **29**:785, 1946.
8. Borley, W. E.: The scleral resection (eyeball-shortening) operation. Tr. Am. Ophth. Soc., **47**:462, 1949.
9. Kronfeld, P. C., and Pischel, D. K.: Scleral resection operation for retinal detachment; an evaluation of the results. Arch. Ophth., **51**:356, 1954.
10. Weve, H.: Bulbusverkürzung durch Reffung der Sclera. Ophthalmologica, **118**:660, 1949.
11. Lindner, K.: Verkürzung des Augapfels durch Faltung. Wien. klin. Wchschr., **61**:206, 1949.
12. Dellaporta, A.: Experimental studies on scleral buckling operation. Am. J. Ophth., **42**:189, 1956.
13. ———: Die experimentelle Verkürzung des Bulbus durch Faltung der Sclera. Klin. Monatsbl. f. Augenh., **119**:313, 1951.
14. Shapland, C. D.: Scleral resection—lamellar. Tr. Ophth. Soc. U. Kingdom, **71**:29, 1951.
15. Paufique, L., and Hugonnier, R.: Traitement du décollement de la rétine par la résection sclérale; technique personnelle, indications et resultats. Bull. et Mém. Soc franç. Ophth., **64**:435, 1951.
16. Berliner, M. L.: Lamellar resection of sclera in the treatment of retinal detachment. Arch. Ophth., **48**:596, 1952.
17. Symposium: Retinal detachment. New York Soc. Clin. Ophth., Jan. 17, 1955. Am. J. Ophth., **41**:698, 1956.
18. Chamlin, M., and Rubner, K.: Lamellar undermining; a preliminary report on a technique of scleral buckling for retinal detachment. Am. J. Ophth., **41**:633, 1956.
19. Everett, W. G.: A new scleral shortening operation, preliminary report. Arch. Ophth., **53**:865, 1955.
20. Castroviejo, R.: New clips and clip-applying forceps for scleral shortening procedures. Tr. Am. Acad. Ophth., **60**:483, 1956.
21. Everett, W. B.: An experimental evaluation of scleral shortening operation. Arch. Ophth., **56**:34, 1956.
22. Shafer, D. M.: Vitreous implant in retinal detachment; a study of 50 consecutive vitreous operations. New York State J. Med., **56**:3300, 1956.
23. Symposium: Retinal detachment; Diagnostic and prognostic factors as found in preoperative examination. Tr. Am. Acad. Ophth., **56**:370, 1952.
24. Dellaporta, A.: Experimental studies on scleral buckling operation. Am. J. Ophth., **42**:189, 1956.
25. Arruga, H.: Personal communication.
26. Shipman, J. S.: Personal communication.
27. Picó, G.: Retinal detachment; postoperative care. In press.

INHIBITION IN STRABISMUS AND A NEW METHOD OF TREATMENT*

W. Isola, M.D., and Berta Stark, M.D.
Montevideo, Uruguay

The purpose of this communication is to report a simple procedure which we have found valuable in breaking up the inhibitions of a visual image in patients with strabismus.

The fact that normal eyes work harmoniously to effect binocular vision forces one to postulate the existence of some mechanisms which keep the eyes "associated." It has been known since Mueller's time that the two maculas, the points of sharpest vision in the retinas, are likewise the points for the best accomplishment of binocular vision—in other words, are corresponding retinal points. It is also generally believed that in the normal individual with parallel visual areas, there is similar correspondence between the perimacular and peripheral retinal areas of the two eyes. On this basis of normal retinal correspondence, one explains diplopia on the assumption that "the same image falls on two retinal points which do not correspond." Even though there are conflicting opinions, such as the theory of the areas of Pannum, nevertheless the theory of normal retinal correspondence is the one most generally accepted to explain binocular vision.

Duke-Elder specifies that the patient with strabismus may choose among the following possibilities:

1. May accept the strabismus as a psychologic diplopia.
2. May suppress one visual image, which is the case in the majority of such patients.
3. May adjust the visual axis, as in heterophorias and convergence insufficiencies.
4. May develop anomalous retinal correspondence, as do many nonconcomitant strabismus patients.

Thus inhibition and the development of anomalous retinal correspondence are the principal ways chosen by the strabismus patient to eliminate diplopia.

Inhibition, neutralization, and suppression are terms of equal value and they represent the same phenomenon. It was Javal who, speaking of neutralization, defined it as an "inhibitory act that permits us to ignore certain disagreeable impressions in vision, and especially unfavorable impressions in binocular vision." Despite various theories and suggestions, the mechanism, the nature, the development, and the locus of inhibition are still unknown. It is recognized, however, that inhibition of a visual impression may be a fundamental part in the act of normal vision, especially in the stage of perceptual integration of images (Sherrington). In ocular dominance, the visual image of the nondominant eye may confuse rather than reinforce the visual impression and so be inhibited. It is also recognized that in the suppression of the so-called physiologic diplopia and in peripheral diplopia one visual image is inhibited. However, these examples of suppression of an image in normal binocular vision do not give information on the mechanism or the locus of the phenomenon. Further, it is possible that these examples of physiologic inhibition in normal binocular vision may not be analogous to the visual inhibition in strabismus.

Even if one accepts the premise that the mechanism of normal physiologic inhibition and that of individuals with strabismus is the same, this does not tell the locus of the phenomenon. Tschermak, Bielschowsky, Braun, Duke-Elder, and many others believe the phenomenon is a psychic process. Harms and others believe the site of the inhibition is peripheral and begins in the ganglion cells of the retina although they admit that the hypothesized supercortical "centers of

* From the Department of Ophthalmology of the University of Uruguay Medical School.

sensorial fusion" are probably the place where the "union of the impressions that produce binocular vision" take place, and that from these centers there arise the inhibitory impulses which are conducted to the periphery by centrifugal fibers.

Whatever may be the mechanism, nature, development, and locus of visual inhibition, in the daily clinic where many strabismus patients are treated, it is a very difficult phenomenon to overcome. The loss of time, which may extend over months, frequently results in abandoning orthoptic training and may occasionally result in premature operation for the strabismus. In the orthoptic clinic of the University of Uruguay Hospital in Montevideo, we have found the following simple procedure most useful in overcoming the inhibition of strabismus.

Procedure for the suppression of inhibition

The procedure which we have found for the suppression of inhibition, be it peripheric, paramacular, or macular, is to instill drops of physiologic salt solution in the eye with the suppression 30 seconds before initiating the orthoptic work. The suppression of the inhibition is very rapid, before there is any time for the drugs to exert any specific action.

The drops are given in the same way as a collyrium; neither the temperature nor the number of drops instilled appear to have any influence.

This simple procedure was always successful. The results in 20 patients with different types of strabismus may be summed up in the following manner:

In 19 patients there was paramacular and macular suppression. In one case the inhibition was in the peripheral retina. Esotropia was present in 14 of these 20 patients. All patients were seen several times. In some of them the suppression of the inhibition remained permanent, that is, persisting after the exercises with the amblyoscope.

In these patients there was anomalous correspondence in 13 cases determined on the amblyoscope. In two of these cases normal retinal correspondence was obtained by this simple procedure.

In a second series of 50 strabismus patients, this same procedure was followed, with the exception that one-percent pilocarpine solution was substituted for the normal saline. The same inhibition of suppression was observed, and it was our impression that this beneficial effect came on more quickly and was somewhat more permanent when pilocarpine was used.

Conclusions and Summary

In all 20 patients with peripheric and macular inhibition treated to date, the inhibition was overcome by the simple procedure of placing normal saline in the conjunctival sac immediately before orthoptic exercises.

The suppression of the inhibition is so rapid that it can only be explained as a reflexive nervous mechanism.

If this is correct, it makes one doubt the existence of corresponding retinal points or corresponding retinal zones. It is easier to explain this sudden suppression of inhibition on the assumption of a central subcortical zone of visual projection, which is subject to reflex nervous impulses.

We believe that the technique here reported to eliminate the inhibition of visual impulses presents a real step forward in the technique for treatment of strabismus.

Department of Ophthalmology, University of Uruguay Medical School.

THE WILLS EYE HOSPITAL

The 125th anniversary of its founding

Irving H. Leopold, M.D.
Philadelphia, Pennsylvania

This year a great hospital is celebrating the 125th anniversary of its founding. In Philadelphia, on April 2, 1832, the cornerstone was laid for the first hospital in the United States devoted solely and completely to the care of the human eye. And now, 125 years later, the Wills Eye Hospital is the largest hospital of its kind in America.

Milestone anniversaries are the traditional times for examination and evaluation —appraisals and reappraisals. Under the powerful and uncompromising lens of the microscope of self-scrutiny, the record of the Wills Eye Hospital has made everyone associated with it, and the city that supports it, both proud and grateful. Today's resident at the Wills, as he takes a long backward look into the history of the hospital, cannot help but be inspired by the achievements of the physicians who have preceded him. He is training at an institution which initiated, developed, and perfected operative techniques and medical treatments which have been adopted throughout the world as standard procedures in the care of the human eye.

In 1831, the city administration in Philadelphia passed an ordinance permitting the city to administer the estate of James Wills, Jr., a wealthy Philadelphia Quaker who had left almost his entire fortune for the establishment of "The Wills Hospital for the Indigent Blind and Lame." For $20,000.00 a lot was purchased extending from Sassafras Street (now Race Street) to Cherry Street, and from Schuylkill Fourth (now 19th) to Schuylkill Fifth (18th Street). The cornerstone was laid April 2, 1832, and the hospital was opened on March 3, 1834 (fig. 1).

Fig. 1 (Leopold). Wills Eye Hospital: The original building.

Appointed this first year, Isaac Parrish, Squier Littell, Isaac Hays, and George Fox were the first attending surgeons at the Wills Hospital, the first of a list that now numbers 59 (table 1). All four of these surgeons were men of unusual attainments, and many of their accomplishments have never been excelled in Philadelphia medicine. Succeeding the first staff came a group of general surgeons, most of whom were on the staff of the Pennsylvania Hospital and used the Wills Hospital to perfect their eye surgery. All of these men were able, if not superior, ophthalmic surgeons.

By 1860 in Philadelphia, the practice of general surgeons devoting some of their time to ophthalmology was becoming a thing of the past, and ophthalmology as a specialty was developing into a science on a firm basis of its own. Partly responsible was the discovery of the ophthalmoscope in 1851. Also about this time came the tremendous contributions to ophthalmology of Albrecht von Graefe, Von Helmholtz and Donders, and the use of general anesthesia. Modern ophthalmology was now a separate branch

TABLE 1

ATTENDING SURGEONS: WILLS EYE HOSPITAL

Name	*Date*	*Name*	*Date*	*Name*	*Date*
Isaac Parrish	1834–1852	William Thomson	1872–1877	J. Milton Griscom	1917–1943
Squier Littell	1834–1864	A. Douglas Hall	1872–1893	Frank C. Parker	1919–1949
Isaac Hays	1834–1854	Richard Levis	1872–1874	Thomas B. Holloway	1919–1924
George Fox	1834–1849	George Strawbridge	1873–1890	B. F. Baer, Jr.	1924–1938
John Neill	1849–1852	Henry S. Schell	1877–1890	Thomas A. O'Brien	1924–1941
Edward Hartshorne	1852–1859	Frank Fisher	1890–1916	Leighton F. Appleman	1924–1939
F. W. Sargent	1852–1857	Charles A. Oliver	1890–1911	Francis Heed Adler	1933–1936
Addinell Hewson	1854–1861	Samuel D. Risley	1890–1917	Louis Lehrfeld	1937–1952
William Hunt	1857–1864	Edward Jackson	1890–1898	Warren S. Reese	1938–1957
Thomas G. Morton	1859–1864	Conrad Berens	1893–1914	Carroll R. Mullen	1939–
George C. Harlan	1861–1864	William Thomson	1896–1902	James S. Shipman	1939–
A. Douglass Hall	1864–1872	John W. Croskey	1897–1900	Edmund B. Spaeth	1939–
Richard J. Levis	1864–1872	P. N. K. Schwenk	1898–1924	William J. Harrison	1939–1953
D. Hayes Agnew	1768–1868	McCluney Radcliffe	1901–1924	Isaac S. Tassman	1939–
George C. Harlan	1868–1901	S. Lewis Ziegler	1901–1916	Wilfred E. Fry	1943–
Ezra Dyer	1872–1873	William Zentmayer	1901–1928	P. Robb McDonald	1949–
H. Ernest Goodman	1872–1896	Wm. Campbell Posey	1902–1919	Irving H. Leopold	1952–
Peter D. Keyser	1872–1897	Paul J. Pontius	1907–1932	Patrick J. Kennedy	1953–
William F. Norris	1872–1907	William M. Sweet	1911–1919	Edward J. Donnelly	1957–
William W. McClure	1872–1901	Burton Chance	1916–1933		

of medical science, and the first full-time specialists were elected to the Wills Hospital staff.

After the Civil War (every member of the Wills Staff saw some active service) the growth and development of the Wills was rapid. Its management was now vested in a newly created Board of Directors of City Trusts, its staff was enlarged, and the contributions of its members to medical literature was impressive.

The creation of the American Ophthalmological Society in 1864 gave impetus to the formation of other ophthalmological societies. Several men, at one time associated with the Wills Eye Hospital, have been president of the American Ophthalmological Society. These include William Norris (1885-1889), Dr. Samuel Risley (1908), Dr. Edward Jackson (1912), Dr. George E. deSchweinitz (1916), Dr. William M. Sweet (1922), Dr. William Zentmayer (1927), Dr. William Campbell Posey (1930), and Dr. Thomas Holloway (1933). The Section on Ophthalmology of the College of Physicians of Philadelphia was organized in 1870. In 1878 the Section on Ophthalmology of the American Medical Association was initiated, headed by Dr. Herman Knapp of New York. In succeeding years, at least six members of the Wills Hospital staff have filled this position. The American Academy of Ophthalmology and Otolaryngology was organized in 1896. Its first president was Dr. Edward Jackson, once a member of the Wills staff.

During the period of from 1870 to 1900 graduate schools in medicine began to be established in America, and a member of the Wills Staff, Dr. R. G. Levis, was the first president of the Philadelphia Polyclinic and College for Graduates in Medicine, founded in 1883. Courses of three months were demanded of each student, and the essentials in ophthalmology were fully covered in that period. Part of this graduate work was carried on at the Wills Hospital. Subsequently, the school was merged with the University of Pennsylvania and became the nucleus of its Graduate School of Medicine.

As an adjunct for clinical teaching, probably no other hospital in America has played a more important part or exerted greater influence in the teaching of ophthalmology than the Wills. Lectures were begun there as early as 1839, instigated by Isaac Parrish. Dr. Fox delivered clinical lectures until his retirement in 1854. In 1867 the board

THE WILLS OPHTHALMIC HOSPITAL,

Race Street, between Eighteenth and Nineteenth Sts., Philadelphia.

A Course of Lectures, Didactic and Clinical, on Ophthalmic Surgery, will be given at the Hospital during the months of November, December, and January, on Saturday evenings, between 8 and 10 o'clock.

The course will embrace all of the important branches of Ophthalmic Science, and will include the *Anatomy and Pathology of the Eye, the Physiology of Vision, the Refraction and Accommodation of the Eye, the Use of the Ophthalmoscope, and the Operative Surgery of the Eye.*

The large Clinics of the Hospital will afford abundant opportunities for the demonstration of the *General Diseases, Optical Defects, and Operative Surgery of the Eye.*

Each member of the class will be afforded instruction in the Use of the Ophthalmoscope, and in the practice of Operations on the Cadaver.

The Diagnosis of the Optical Defects which produce *Long, Short, or Weak Sight, Astigmatism, Strabismus, etc.*, and their Correction by the Scientific Use of Glasses, will be illustrated by apparatus and Clinical demonstration.

Fee for the Course . . . . . Ten Dollars.

Operative and Clinical Surgery of the Eye.
R. J. Levis, M. D., N. W. cor. Arch and 13th Sts.

Anatomy of the Eye, and Ophthalmoscopy.
Geo. C. Harlan, M.D., 1806 Chestnut St.

Physiology of Vision, Refraction, and General Diseases of the Eye.
Ezra Dyer, M.D., 1429 Walnut St.

PRACTICAL OPHTHALMOSCOPY.

EVENING DEMONSTRATIONS WITH THE OPHTHALMOSCOPE.

Wills Hospital, Race above 18th Street, Phila.

A Fifth Course will begin early in April, in the Operating-Room of the Hospital, and will be continued weekly until the first of July.

It will be the aim of the instructor to give the student a working knowledge of the use of the Ophthalmoscope in the Diagnosis of Diseases of the Internal Eye, and with this view especial care will be taken that the observations are verified by each member of the class.

The special studies will be the Natural and Morbid Appearances of the Optic Nerve, the Retina, Choroid, Vitreous Humor, and the various degrees of Lenticular Opacity.

For illustration and comparison the best and most recent foreign colored drawings are provided. Apply to A. D. HALL, M. D., *one of the Surgeons to Wills Hospital*, 1623 Spruce Street.

Fee Ten Dollars.

Fig. 2 (Leopold). Advertisement of early lectures in the Wills Eye Hospital, appearing in the *American Journal of Medical Sciences*, in 1869 and in 1873.

passed a by-law to continue lectures of the staff to visiting physicians and students. Appearing as an advertisement in the *American Journal of the Medical Sciences* were the courses of lectures at the Wills Hospital given by Dr. Hall. In 1873 similar courses were advertised by Dr. Levis, Dr. Hall, and Dr. Dyer.

Residents at the Wills were first designated as medical residents, later as resident surgeons. There was only one resident surgeon at a time until 1892, when the number was increased to two. This was increased to three in 1914, to four in 1924, to five in 1928, to six in 1931, and at the present time there are 14. The term of service was one year until 1867, when it was made six months; in 1870 it was again made one year; in 1915 it was made 18 months, in 1924, 16 months, and in 1928, 17 and a half months. It is now a two-year residency. To date 289 residents have been trained.

The literature devoted to ophthalmology during the 125 years of the existence of the Wills Hospital has been enormous. The first journal devoted largely to ophthalmology was the *Journal für Chirugie und Augenheilkunde*, published in Germany in 1820 by Carl Ferdinand von Graefe and Philip von Walther, but publication lasted only 20 years. The *Annales d'Oculistique*, edited by Cunier of Brussels in 1838, is still in existence and is the oldest eye journal in the world. The first American publication was The American Journal of Ophthalmology, published in 1854. The *Archives of Ophthalmology* appeared in 1869, the *Ophthalmic Record* in 1891, the *Annals of Oph-*

Fig. 3 (Leopold). Wills Eye Hospital: The present building.

thalmology in 1892. In 1918, Dr. Edward Jackson of Denver, Colorado, a former surgeon at the Wills, amalgamated six ophthalmologic publications and founded the present third series of THE AMERICAN JOURNAL OF OPHTHALMOLOGY. After the death of Herman Knapp in 1900, the *Archives of Ophthalmology* was continued by his son, Arnold Knapp, and taken over by the American Medical Association in 1928. Dr. William Zentmayer, emeritus attending surgeon of the Wills staff is an editor, and Dr. Francis Heed Adler, formerly attending surgeon of the Wills Hospital staff and now consultant to the hospital, is editor-in-chief of the *Archives of Ophthalmology*. He is presently professor of ophthalmology at the School of Medicine of the University of Pennsylvania.

By 1930 the old Wills Hospital building was no longer adequate. During the one year of 1930 there were 24,940 cases treated there. Accordingly, in June, 1930, the entire property was sold to Mr. Cyrus H. K. Curtis for $1,100,000 and in March, 1931, ground was acquired at the northwest corner of 16th and Spring Garden Streets, the present site of the Wills Eye Hospital.

The present Wills Eye Hospital built in 1932 (fig. 3) has 244 beds, all for ocular patients only. In 1932 private facilities were included in the new buildings so that the attending staff would be able to concentrate its efforts at the institution. Additional private rooms were addded in 1955, as the nurses moved from the hospital into their own apartment building adjacent to the hospital.

The first hospital could accommodate 20 patients, and the dispensary service was started in 1839. Wills Hospital now sees approximately 14,663 out-patients (in addition to nearly 11,000 emergency cases) and 5,144 in-patients each year. In 1956, 87,904 out-patient visits were made and 60,339 in-patient days of service were rendered; 5,386 operations were performed.

The staff now includes 85 ophthalmologists and 29 ancillary medical personnel. The patients in this institution have available for their needs consultants in all branches of medicine. The institution has its own department of internal medicine and its subdivision of cardiology, allergy, immunology, hematology, and so forth; department of pediatrics, department of dermatology, radiology, otorhinolaryngology, and neurology. Residents and fellows from other institutions in the area spend considerable time at the Wills Eye Hospital learning

neuro-ophthalmology and ocular problems associated with dermatology.

Students have used these facilities almost since its inception. At present the medical students of Jefferson Medical College are taught clinical ophthalmology here and the ophthalmic student physicians of the Graduate School of Medicine of the University of Pennsylvania spend many hours each week under the guidance of the staff, seeing and learning from the tremendous number of ocular problems encountered.

At all times the teaching members of the Wills Hospital staff have attempted to fulfill their duty to the student but also take care of and safeguard the interests of the patient. It is evident that, although this institution commenced mainly for the care of the visually impaired patient, it has become over the years an important teaching hospital as well.

It became apparent that this institution that had contributed so much to the care of the visually handicapped patients and to the clinical teaching of ophthalmology for so many years had much to offer in the field of clinical and basic research. Research work done in close proximity to teaching wards and clinics offers mutual stimulation to clinician, patient, student, and research scientist. Laboratories of clinical research should be associated with hospitals, because patients are required for these studies, and it is here, not in medical school buildings, that one finds patients. A patient benefits, for he stands his best chance to receive help in a clinic and hospital where the illness from which he is suffering is under intensive research study. Patients today are aware of this and seek out institutions that offer this combined type of facility. Research improves the quality of care the patient receives.

For these reasons, a research department under the direction of Dr. Irving H. Leopold was started in 1949 and a research laboratory devoted to investigations in ophthalmic dysfunctions was formally dedicated

Fig. 4 (Leopold). Wills Eye Hospital: The research laboratory.

in 1950 (fig. 4). This now consists of departments of ocular biochemistry, microbiology, pathology, and physiology. The research staff includes 20 full-time investigators.

For many years the Wills Hospital has been a training ground for ophthalmic nursing. The nurses trained here are all graduate nurses from other institutions all over the world.

Individuals who have received all or some of their ophthalmic training at the Wills Hospital have joined medical school staffs throughout the United States and the world. Many of these have become the heads of the Departments of Ophthalmology in medical schools so that directly and indirectly this institution has and continues to contribute in a considerable degree to ophthalmic training.

Summary

In 1832 a hospital was started that has since taken care of millions of patients with ocular problems; has trained nurses, residents, and teachers in ophthalmology, as well as contributed new observations, techniques, therapies, and diagnostic measures.

The hospital has existed for 125 years, through four wars, and through many changes in the concepts of medical care, training, and administration. No institution can exist for 125 years without having stim-

ulated some criticism of its administration. At some crossroad of its journey or in looking backward, the Wills Hospital has had its share of this. However, it is impossible to overestimate the great influence of this institution on ophthalmology during the last century.

Theories of medical practice, teaching, and administration change, and the Wills Hospital will also change, as it has in the past, always guided by its aim of providing the best possible care for the patients with ocular problems and the optimal training of ophthalmic physicians and surgeons not only now but in future generations.

1601 Spring Garden Street (31).

PARTIAL LAMELLAR CORNEAL GRAFTS IN RABBITS*

Histologic observations on the survival of the stromal cells of the lamellar corneal graft

Walter Kornblueth, M.D. and Edith Nelken, M.D.
Jerusalem, Israel

Very few histologic studies[1,2] on experimental lamellar grafts have been reported since the work of Bonnefon and Lacoste[3] on rabbits (1913). The lack of a suitable surgical technique has apparently been the reason for the scarcity of such experiments. Bonnefon and Lacoste insisted that nearly all the fixed cells of a lamellar graft die within a short period of grafting and on the third day the graft contained only necrotic elements. This necrosis is followed by cellular regeneration from keratoblasts.

From earlier histologic work on perforating grafts[4] it seemed improbable that in a partial lamellar graft all the stromal cells should die within a short time. The present study was undertaken to prove that a great part of the stromal cells in a lamellar graft survive for a long period. The question of their eventual replacement from cells of the host remains still open. In order to collect satisfactory material for histologic study a surgical technique had to be developed which gave a high proportion of clear grafts.

Method

Rabbits from various stocks weighing about 2.0 kg. were used. They were operated in pairs and the corneal discs cut with a trephine were exchanged. Intravenous nembutal was used as general anesthetic and drops of two-percent pantocaine for topical anesthesia. Hair around the eye was trimmed and the shaved area swabbed with soap and water. The eye was proptosed and kept in this position by putting traction sutures through two opposite recti muscles.

A lamellar incision was made with a Franceschetti trephine 7.1 mm. in diameter, the guard having been set at 0.3-mm. depth. The lamellar incision to the desired depth (one-half to two-thirds or the thickness of the cornea) was made without removing the trephine.

A traction suture was placed through the free margin of the disc. Slight traction was produced with the suture and the disc dissected with a Bard Parker No. 15 blade. One corneal disc was exchanged immediately with another one which had been prepared simultaneously in the same manner by a second surgeon. The transplant was held in

* From the Department of Ophthalmology, Rothschild-Hadassah-University Hospital.

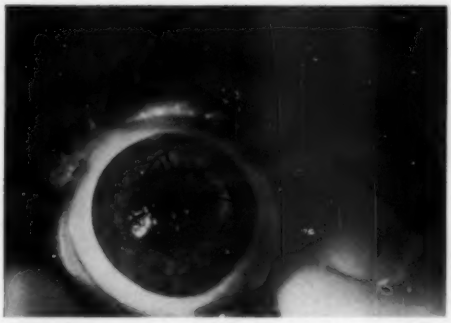


Fig. 1 (Kornblueth and Nelken). Lamellar corneal graft held in place by a continuous criss-cross suture (24 hours after operation).

place by a continuous criss-cross corneal suture inserted into the recipient cornea as closely as possible to the edge of the graft (fig. 1). The lids were tied by one suture.

No antibiotic was used following the operation except in rare cases with copious secretion when streptomycin drops (one-percent solution) were applied. The eyes were cleaned daily with cotton. The lid and corneal sutures were removed after seven days.

Results

Partial lamellar corneal grafts were performed on 150 eyes in 90 rabbits. In the first 94 eyes (60 rabbits) successful clear grafts were obtained in only 21 percent. As the surgical technique improved the percentage of successful clear grafts in the last series of 52 eyes (30 rabbits) rose to 55 percent.

During the first two or three days it was frequently difficult to predict which graft would stay clear or which would become cloudy. As a rule a successful graft showed only slight edema during the first three days which subsided gradually within seven to 10 days. In grafts that eventually became cloudy edema was somewhat more pronounced. All the grafts which were clear for seven to 10 days postoperatively remained clear for the whole period of observation (up to seven months). On slitlamp examina-

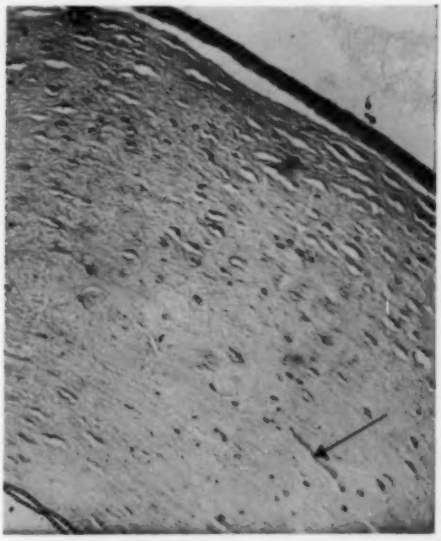


Fig. 2b (Kornblueth and Nelken). Section of lamellar corneal graft (12 hours after operation). Slight edema of the graft. Epithelial and stromal cells of the graft preserved. (Hematoxylin-eosin, ×125.) ↓ Junction between graft and recipient cornea.

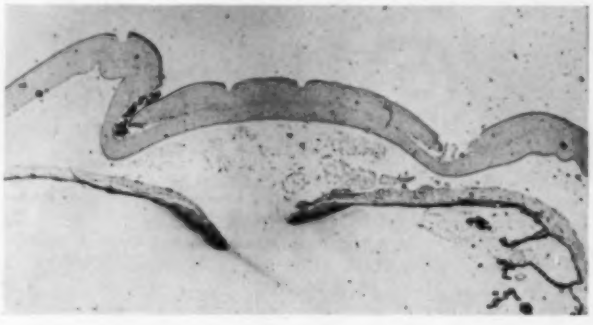


Fig. 2a (Kornblueth and Nelken). Section of lamellar corneal graft (12 hours after operation). Graft well atttached to underlying recipient cornea. (Hematoxylin-eosin, ×18.)

Fig. 3 (Kornblueth and Nelken). Section of lamellar corneal graft (24 hours after operation). Slight edema of the graft. Epithelial and stromal cells of the graft preserved. (Hematoxylin-eosin, ×130.) ↓ Junction between graft and recipient cornea.

tion all the recipient corneas showed superficial blood vessels which reached the wound edges by the seventh postoperative day, some of which later invaded the edge of the graft.

Histologic examinations were made on clear grafts 12, 24, and 36 hours postoperatively and from then on at daily intervals up to one week, at weekly intervals up to two months and at monthly intervals up to seven months.

No detailed description on the various histologic changes taking place in the graft and the recipient cornea will be given as this was discussed thoroughly in the work of Bonnefon and Lacoste.[3] The present report deals mainly with the fate of the stromal cells of the graft.

There was slight edema of the stroma of the graft which subsided gradually after seven to 10 days. During the whole period of observation most of the stromal cells of the graft appeared normal and did not show any signs of degeneration. No abnormal acellular areas were seen except near the wound edges, where the graft as well as the

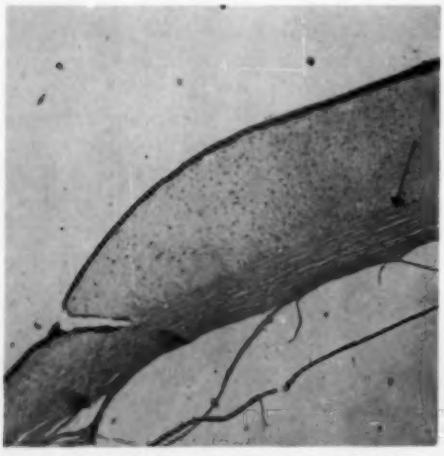


Fig. 4a (Kornblueth and Nelken). Section of lamellar corneal graft (36 hours after operation). Epithelial growth over the wound edges. Scarcity of stromal cells of host and recipient cornea near wound edges. (Hematoxylin-eosin, ×44.) ↓ Junction between graft and recipient cornea.

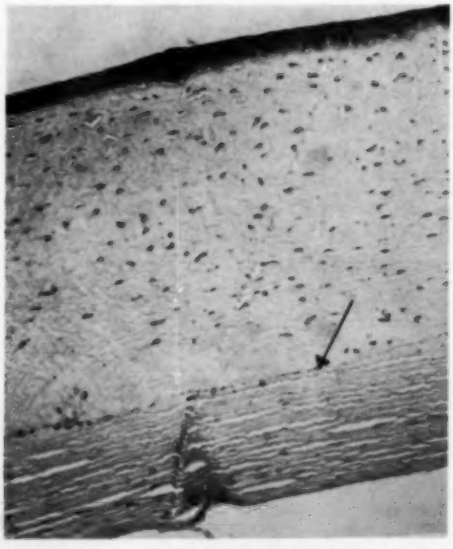


Fig. 4b (Kornblueth and Nelken). Section of lamellar corneal graft (36 hours after operation). Slight edema of the graft. Epithelial and stromal cells of the graft preserved. (Hematoxylin-eosin, ×100.) ↓ Junction between recipient graft and cornea.

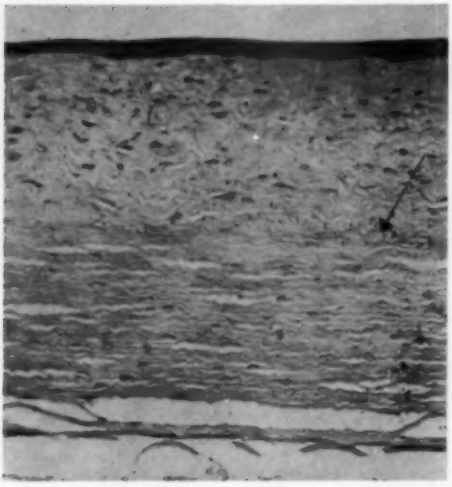


Fig. 5 (Kornblueth and Nelken). Section of lamellar corneal graft (two days after operation). Slight edema of the graft. Epithelial and stromal cells of the graft preserved. (Hematoxylin-eosin, ×135.) ↓ Junction between graft and recipient cornea.

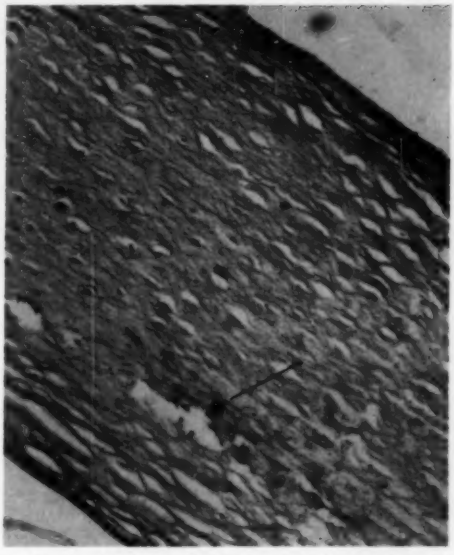


Fig. 7 (Kornblueth and Nelken). Section of lamellar corneal graft (four days after operation). Slight edema of graft. Epithelial and stromal cells of graft preserved. (Hematoxylin-eosin, ×135.) ↓ Junction between graft and recipient cornea.

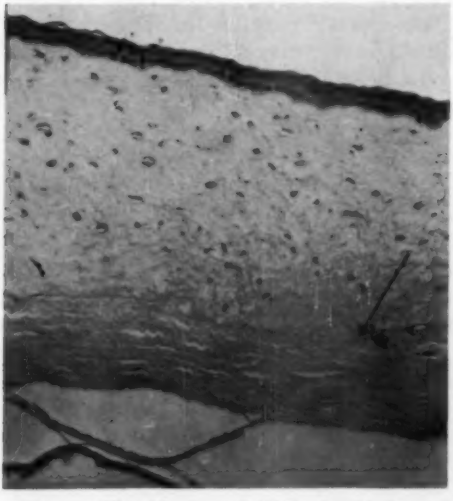


Fig. 6 (Kornblueth and Nelken). Section of lamellar corneal graft (three days after operation). Slight edema of the graft. Epithelial and stromal cells of the graft preserved. (Hematoxylin-eosin, ×145.) ↓ Junction between graft and recipient cornea.

Fig. 8 (Kornblueth and Nelken). Section of lamellar corneal graft (seven days after operation). Slight edema of graft. Epithelial and stromal cells preserved. (Hematoxylin-eosin, ×150.) ↓ Junction between graft and recipient cornea.

Fig. 9 (Kornblueth and Nelken). Section of lamellar corneal graft (13 days after operation). Slight edema of graft. Epithelial and stromal cells preserved. (Hematoxylin-eosin, ×125.) ↓ Junction between graft and recipient cornea.

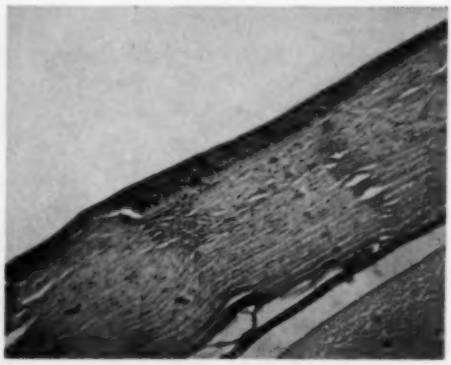


Fig. 10 (Kornblueth and Nelken). Section of lamellar corneal graft (two months after operation). Irregularity of connective tissue fibers at wound edge. No difference between corneal graft and recipient cornea visible. (Hematoxylin-eosin, ×100.)

recipient cornea were poor in cells during the first postoperative week (figs. 2a, 2b, 3, 4a, 4b, 5, 6, 7, 8, 9, and 10). Mitotic figures were occasionally observed in the stromal cells of the graft and of the recipient cornea adjacent to the wound edges from the third to the seventh day after the operation (figs. 11a and 11b). A small number of polymorphonuclear leukocytes invaded the stroma of the graft, especially the areas near the wound edges.

The epithelium of the graft appeared to persist. The number of the preserved layers was depending on the handling of the epithelium of the graft during the operation. Mitotic figures were found in the basal layer

Fig. 11a (Kornblueth and Nelken). Section of lamellar corneal graft (five days after operation). Mitosis in stromal cell of graft. (Hematoxylin, ×160.)

Fig. 11b (Kornblueth and Nelken). Same section as 11a, higher magnification of marked area. (Hematoxylin, ×1,600.)

as early as 24 hours after grafting (figs. 12a and 12b).

Cloudy grafts showed lack of adherence to the recipient bed and a very heavy infiltration with polymorphonuclear leukocytes with destruction of the stromal cells (fig. 13).

Discussion

The work of Bonnefon and Lacoste performed in 1913 is the only thorough experimental study on the histologic changes in partial lamellar autologous grafts. These authors stated that the most striking finding in the first few days after grafting was the total necrosis of the original fixed stromal cells of the graft.

Offret,[5] discussing the work of Bonnefon and Lacoste, thought that the chances of survival of the original stromal cells of a graft are greater in grafts cut by a trephine which attained a thickness of two-thirds to three-fourths of the stroma, than in thin and superficial grafts cut by a corneal knife as practiced by Bonnefon and Lacoste. Choyce[1] examined histologically partial lamellar grafts in rabbits three and one-half weeks,

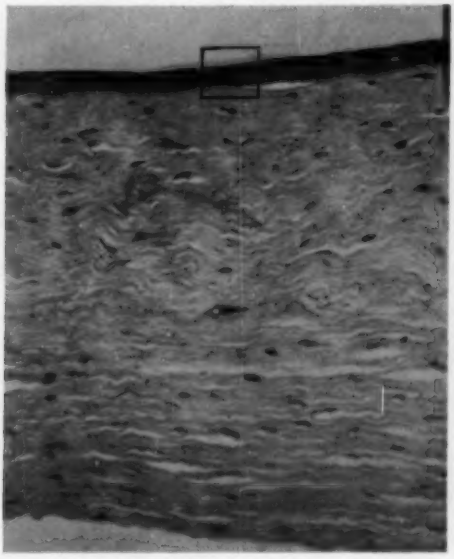


Fig. 12a (Kornblueth and Nelken). Section of lamellar corneal graft (two days after operation). Mitosis of epithelial cell of graft. (Hematoxylin-eosin, ×200.)

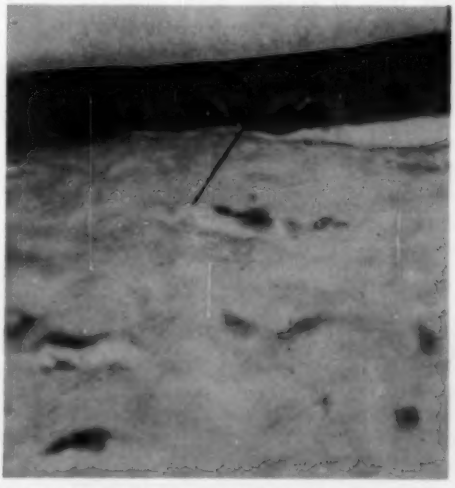


Fig. 12b (Kornblueth and Nelken). Same section as 12a, higher magnification of marked area. (Hematoxylin-eosin, ×580.)

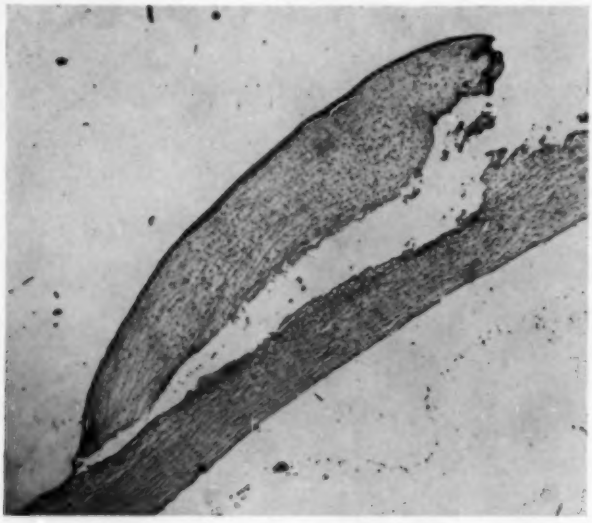


Fig. 13 (Kornblueth and Nelken). Section of cloudy lamellar corneal graft (24 hours after operation). Lack of adherence of the graft to the recipient cornea. Heavy infiltration of graft with polymorphonuclear leuckocytes. (Hematoxylin-eosin, ×33.)

five and seven months after operation and found no signs of significant necrosis or replacement of the grafted tissue. However, these histologic studies were done too late to detect necrosis of the stromal cells of the graft which might have occurred shortly after the operation.

The present study showed clearly that a very great proportion of the stromal cells of the graft survived. At no time was there a massive destruction of the stromal cells and the only areas poor in cells were found near the wound edges where the stromal cells were damaged by the trephine blade. The most important step for surgical success seemed to be the immediate exchange of the grafts after they were removed from the cornea. This was accomplished by timing the removal of the graft performed simultaneously by two surgeons.

Summary

A surgical technique for partial lamellar grafts in rabbits which gave up to 55 percent clear grafts is described. Histologic examination of lamellar grafts from as early as 12 hours and up to seven months after transplantation showed that the great majority of the stromal cells survived. At no time was there massive destruction of the stromal cells of the graft, as has been described following other surgical techniques.

P.O. Box 499.

References

1. Choyce, D. P.: Nonperforating keratoplasties in rabbits. Tr. Ophth. Soc. U. Kingdom, **69**:55, 1949.
2. Shafer, D. M.: Experimental corneal grafts of the lamellar partial type. Am. J. Ophth., **33**:26 (June, Pt. II) 1950.
3. Bonnefon, G., and Lacoste, A.: Recherches histologiques sur la greffe corneenne autoplastique. Arch. d'Ophtal., **33**:206, 1913.
4. Maumenee, A. E., and Kornblueth, W.: Symposium: Corneal transplantations: IV. Physiopathology. Am. J. Ophth., **31**:1348, 1948.
5. Offret, G.: The histopathology of the corneal graft. Corneal Grafts. London, Butterworth, 1955.

CONGENITAL RETINAL DYSFUNCTION*

GÖSTA KARPE, M.D., AND BIRGITTA ZETTERSTRÖM, M.D.
Stockholm, Sweden

An unelucidated eye disease with a special type of bilateral retinal damage, demonstrated by means of electroretinography, is denoted here as congenital retinal dysfunction. By this is meant a congenital condition with bilateral, greatly reduced visual function and a pathologic electroretinogram, but with no definite, objective pathologic findings in the eyes, including the fundus.

CASE MATERIAL

Four cases have been observed at the Eye Clinic of Karolinska Sjukhuset between 1948 and 1957. All four children were brought for ophthalmologic examination because their parents had suspected greatly impaired sight at an early age, or had noticed nystagmus, squinting, or deficient fixation.

No family history of reduced visual acuity or eye disease could be traced, nor was there any consanguinity. None of the children had shown any signs of prematurity; all were born within the calculated term and had a birth weight over 2,800 gm.

Mental retradation of moderate degree has been detected in two of the four children.

OPHTHALMOLOGIC FEATURES AND ELECTRORETINOGRAPHIC INVESTIGATIONS

The media were normally clear. The fundus picture was the same in all the cases, and the features could not be said to differ definitely from the normal ones. Thus in all cases the fundus was deficient in pigment, which had a very faintly uneven distribution, giving the fundus a granular appearance. Single or several small, round yellow spots, which differed only slightly in color from the rest of the fundus, were observed. The discs could never be denoted with certainty as pathologically pale. All these details were so inappreciably developed that they lay within the normal range of variation. The retinal vessels were normal throughout. The only definitely pathologic feature was that the macular reflexes were absent or poorly marked. No signs of retinal detachment or other retinal disease were present.

None of the children was completely blind but all had high-grade impairment of vision.

The ophthalmologic condition was otherwise normal, except for a squint (one child), or nystagmus (three children).

The electroretinogram was recorded with Karpe's[1,2] method (1945 and 1948). The intensity of the stimulus light was 20, 80, and 800 lux. Electroretinographic recordings were made from both eyes.

In normal children, the electroretinogram is entirely lacking at birth, or an extremely small b-potential can occasionally be recorded. After 48 hours, an extremely small b-potential can always be recorded. It increases in size during the first six months of life. At six months, the b-potential is about 0.22 mV. measured from the isoelectric point, that is, the baseline of the curve. At one year, the corresponding figure is about 0.24 mV. The latency and duration of the b-potential, as well as the type of curve, are then in agreement with those in the adult (Zetterström,[3] 1951).

From the developmental point of view, the electroretinogram of the four children in question was greatly retarded as compared to that of the children with healthy eyes. Once an electroretinogram had appeared, its size was also considerably less than that of normal children of corresponding age.

Some parallelism seems to exist between development of the electroretinogram and of visual function. It has been possible to record an improvement in visual function in connection with an increase in size of the b-potential, as compared to that in an earlier examination. The improvement is reliable

* From the Eye Clinic, Karolinska Sjukhuset.

although the visual acuity must be given with some reservations, in view of the relative unreliability of determinations of visual acuity in children.

Table 1 is a survey of the four cases. It gives the age, visual acuity, electroretinogram, and fundus picture at the various examinations.

It can be inferred from the table that, when the electroretinogram could be recorded, it appeared at a considerably later age than in children with normal eyes.

Figure 1 shows the appearance of the electroretinographic curve in one of the children (G. U.) at different ages. For the sake of clarity, a diagram has been made of the size of the b-potential in relation to the age (fig. 2). The mean values of the b-potential in children with healthy eyes are also shown in this diagram for comparison.

TABLE 1

SURVEY OF FOUR CASES OF CONGENITAL RETINAL DYSFUNCTION

Case	Electroretinogram b-potential mV.	Normal Size of b-potential mV.	Age	Visual Acuity	Fundi	Comments
G.U.	RE = extinguished? LE = extinguished	0.15	3 mo.	RE LE = P*	Deficient in pigment. Suggested granularity in peripheral parts of retina. Vessels and discs normal. Macula normal. Nystagmus	Normally developed. Died at 2½ yr. of uremia. Polycystic kidneys
	RE = 0.14 LE = 0.07	0.25	6 mo.	RE LE = P		
	RE = 0.20 LE = 0.20	0.35	1 yr. 4 mo.	RE LE = P		
	RE = 0.17 LE = 0.19	0.35	1 yr. 10 mo.	RE LE = Suff. to get about unaided		
L.H.	RE = extinguished LE = 0.10	0.25	8 mo.	RE LE = P	Deficient in pigment. Vessels and discs normal. Macular reflexes lacking. Nystagmus.	Mentally retarded
	RE = 0.05 LE = 0.10	0.35	2 yr.	RE = hand movements LE = hand movements		
	RE = 0.24 LE = 0.27	0.35	7 yr.	RE LE = 1/60		
	RE = 0.33 LE = 0.30	0.35	8 yr.	RE LE = 3/60		
A.C.	RE = 0.10 LE = 0.10	0.25	8 mo.	RE LE = P	Deficient in pigment. A few small yellow foci in periphery. Vessels and discs normal. Macular reflexes lacking. Conc. conv. squint RE.	Normally developed
	RE = 0.10 LE = 0.10	0.25	1 yr. 1 mo.	RE LE = P		
	RE = 0.12 LE = 0.09	0.35	1 yr. 8 mo.	RE = 2/50 LE = hand movements		
	RE = 0.25 LE = 0.17	0.35	3 yr.	RE = 3/50 LE = 1/50		
	RE = 0.25 LE = 0.20	0.35	4 yr.	RE = 4/50 LE = 1/50		
R.S.	RE LE = extinguished	0.35	6 yr.	RE LE = 1/60	Retina normal. Vessels and discs normal. Macular reflexes lacking. Nystagmus	Mentally retarded
	RE = small suggested LE = neg.	0.35	7 yr.	RE LE = 3/60		

*P = Perception of light.

Discussion

Ocular malformations can be divided into several types, among them those consisting of gross abnormality of structure, showing a disorder of organogenesis present from the beginning, for example, coloboma and aniridia. They may consist of a disorder of differentiation of one tissue only, structure being normal, as in Oguchi's[4] disease and albinism. Other malformations are in the form of postnatal degenerative processes, for example, retinitis pigmentosa. Practically all germinal defects are bilateral, even if the degree of malformation is not the same in both eyes.

It has not yet been established with certainty from which layer or layers of the retina the electroretinogram is generated. Consequently, it is only possible to make hypothetical inferences regarding the site of the primordial anomaly of the retina which seems to exist in the present cases.

A condition denoted as retinal dysplasia or

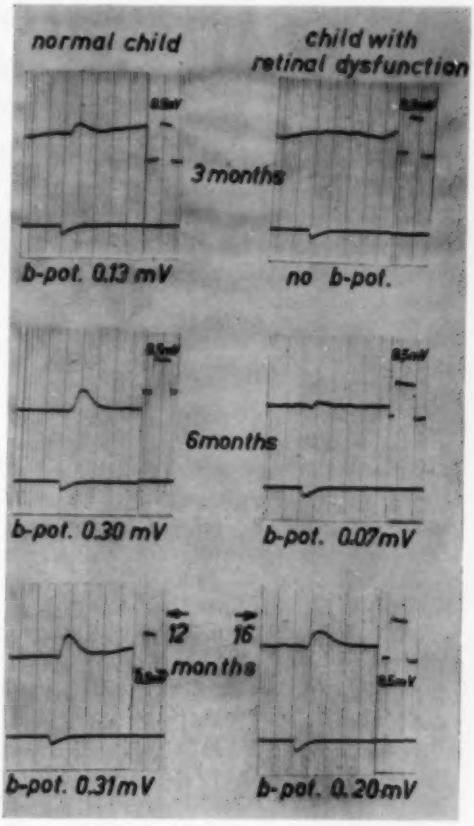


Fig. 1 (Karpe and Zetterström). To the left, the electroretinogram in normal infants at varying ages. To the right, the electroretinogram at corresponding ages in a case of retinal dysfunction (case G. U.).

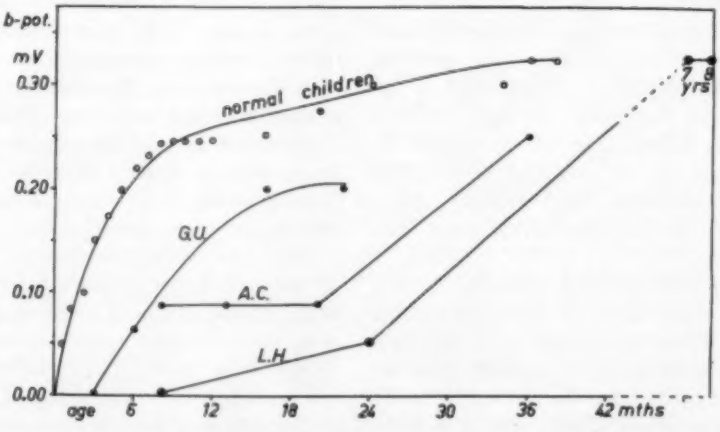


Fig. 2 (Karpe and Zetterström). The b-potential at various ages in normal children and in three cases of retinal dysfunction.

Krause's[5] disease, a severe malformation of both eyes, particularly of the retina, which finally becomes completely detached and produces blindness, presumably cannot be analogous with this disease. Moreover, Krause's disease is associated with encephalodysplasia, and occurs almost exclusively in prematures. It has recently been suggested that this is not a separate disease, but that it is probably retrolental fibroplasia, accompanied in such cases by intracranial anomalies (Mann,[6] 1956). The four cases reported here exhibit no signs whatsoever of retinal dysplasia.

The cases in question seem rather to represent anomalies of the type of albinism, in which the pigmented epithelium of the retina and the rest of the eye is lacking. Another disease to which they seem to bear a resemblance is Oguchi's disease, in which only the light-perceptive elements are malformed from birth. In this condition, objective findings are an inappreciable reduction of visual acuity and congenital hemeralopia.

In albinism, the electroretinogram is normal (Zetterström,[7] 1956). The electroretinogram has been recorded in adults with Oguchi's disease by Hirose[8] (1952) and François, Verriest, and De Rouck[9] (1956). Both found that the b-wave was lacking, contrary to these cases of congenital retinal dysfunction.

The characteristic feature of the degenerative types of malformation, for example, tapetoretinal degeneration and retinitis pigmentosa, is that they undergo constant, continuous exacerbation. In the disease described here, on the contrary, an improvement in visual acuity and a tendency to normalization of the electroretinogram have taken place.

In congenital retinal dysfunction, there is evidently some form of developmental retardation of the retinal elements from which the electroretinogram is elicited. Theoretically, it is conceivable that an electroretinogram can be recorded only when a few elements have reached a certain degree of development, and that when the number of functioning elements subsequently increases, the recordable potential also increases in size. Compared with the conditions in children with healthy eyes, this development would be greatly retarded in the disease in question, and might fail to occur altogether in certain cases.

Without an electroretinographic examination, these children would probably have been classified under the uncertain term of cerebral blindness, in view of the severe amblyopia and lack of definitely pathologic fundus changes. This is in particular since two of them showed signs of brain damage in the form of mental retardation, which is apparent even if their poor sight is taken into account. Moreover, in amblyopia due to visual nerve lesions alone or visual nerve atrophy of descending type, as well as in purely cerebrally conditioned damage, a normal electroretinogram is obtained (Granit and Helme,[10] 1939, Karpe,[1] 1945) in contrast to the present cases.

The possibility nevertheless remains open that brain damage, in addition to the retinal damage demonstrated by the electroretinogram, is also present in these children, and that it contributes to the impairment of vision.

Summary

An account is given of four cases of a hitherto undescribed eye disease, which has been demonstrated by electroretinography and named congenital retinal dysfunction.

All the patients had greatly reduced visual acuity with no definite, objective pathologic findings in the eyes, visual paths, or visual centers.

The electroretinogram was pathologic in all these cases. Development of the electroretinographic potential and visual function is greatly retarded.

Neither a family history of eye disease nor consanguinity could be traced.

The nature of the disease is discussed.

Karolinska Sjukhuset (60).

References

1. Karpe, G.: The basis of clinical electroretinography. Acta ophth., Suppl. 24, 1945.
2. ———: Apparatus and method for clinical recording of the electroretinogram. Doc. Ophth., **2**:268, 1948.
3. Zetterström, B.: The clinical electroretinogram: IV. The electroretinogram in children during the first year of life. Acta ophth., **29**:295, 1951.
4. Oguchi, C.: Ueber die einartige Hemeralopi mit diffuser weissgraulicher Verfärbung des Augenhintergrundes. Arch. f. Ophth., **81**: 109, 1912.
5. Krause, A. C.: Congenital encephalo-ophthalmic dysplasia. Arch. Ophth., **36**:387, 1946.
6. Mann, I.: Developmental Abnormalities of the Eye. London, Cambridge Univ. Press, 1957.
7. Zetterström, B.: Studies on the Postnatal Development of the Electroretinogram in Newborn Infants. Tryckeri AB Thule, 1956.
8. Hirose, T.: Electroretinogram in night-blindness. Acta Soc. ophth. Japan, **56**:732, 1952.
9. François, J., Verriest, G., and De Rouck, A.: La maladie d'Oguchi. Ophthalmologica, **131**:1, 1956.
10. Granit, R., and Helme, T.: Changes in retinal excitability due to polarization and some observations on the relation between the processes in retina and nerve. J. Neurophysiol., **2**:556, 1939.

IMAGE ALTERNATION FOR ANISEIKONIA DETERMINATION*

Gerhard A. Brecher,† M.D., David M. Winters, M.D., and Clark A. Townsend, A.B.

Columbus, Ohio

Introduction

It was the initial aim of the present investigation to study a new approach to aniseikonia measurements which would make it independent of binocular fusion. This approach proved not to be superior to presently used techniques. However, it permitted interesting observations about the central nervous integration of binocular images which plays a role in normal binocular vision as well as in aniseikonia.

Aniseikonia is commonly determined by the use of two techniques, stereoscopic (or "space") and vernier (or "standard") eikonometry, each of which has certain advantages and drawbacks.

The principle of the space eikonometer is based on the effect of the neural integration of incongruent images resulting in a measurable alteration of space perception.[1,2] Adequate binocular fusion and stereopsis of the patient are a prerequisite for the use of this method.

The principle of the standard eikonometer is based on the direct comparison of two monocular images.[3] As in the space eikonometer, accomplishment and maintenance of binocular fusion are also necessary for the use of the standard eikonometer. However, binocular fusion in the standard eikonometer is often rendered difficult by the occurrence of fusion disparities and of competitive fusion impulses arising from the monocular targets.[2,4]

Various suggestions have been made either to improve the stabilization of binocular fusion or abolish it. With the latter procedure the sizes of unfused diplopic or haploscopically presented monocular targets can be compared. However, even when rather dissimilar targets are chosen in order to reduce fusion impulses, there remains enough fusion tendency of the targets to obviate measurements.[4–6] It is, therefore, difficult

* From the Departments of Ophthalmology and Physiology, College of Medicine, The Ohio State University, and the Department of Physiology, Emory University, Atlanta, Georgia. Presented at the meeting of the Association for Research in Ophthalmology, East Central Section, Detroit, Michigan, January 7, 1957.

† For reprints write to Dr. Brecher, Department of Physiology, Emory University, Georgia.

to compare the relative sizes and shapes of two monocularly presented targets when they are viewed simultaneously. Since knowledge of difference between the images of both eyes is not only of some practical importance but is also of interest for an understanding of normal and abnormal binocular fusion processes a simple method was developed which permits an image comparison by successive target presentation.

This method is based on the previously reported observation that identical binocular fusion stimuli can be presented intermittently to both eyes at such slow frequency that fusion does not occur.[7] The aim of comparing the size and shape of the two images is realized in the new method by simply alternating the images of the two eyes slowly enough to prevent binocular fusion but still fast enough for permitting a comparison.

Since with the procedure of "image alternation" the subject would compare directly the images of his two eyes, it was believed that this method might lend itself for aniseikonia determination. This aspect was studied by measuring aniseikonia with the new method.

Apparatus

The experimental arrangement is illustrated in Figure 1. The target for both eyes was a circle of 6.8 cm. diameter, drawn with India ink on white cardboard (60 by 60 cm.) (A and A_1). The cardboard had a brightness of 24 foot-lamberts. The observation distance was one meter and the diameter of the circle subtended four degrees at the nodal point of each eye. The target circle was presented alternately to each eye by viewing it through a rotating sectored disc (B and B_1) close to the eyes. Under such conditions the target was seen by the left eye through one sector (C and C_1) while the right eye was occluded and shortly thereafter the target was seen by the right eye through the other sector (D and D_1) while the left eye was occluded by the opaque area of the disc.

The disc rotated with one revolution every one or two seconds, resulting in exposure times of 0.25 or 0.5 second respectively for each eye and occlusion times of 0.75 or 1.5 seconds respectively for each eye. This permitted comparison of right and left images at an optimal alternation frequency without appreciable disturbance by after-images. Afocal (size) lenses (E) of varying magnification were used to alter the apparent physical dimensions of the target (A). In order to facilitate the discrimination of images, sheets of slightly tinted transparent cellophane were inserted in the cut-out areas

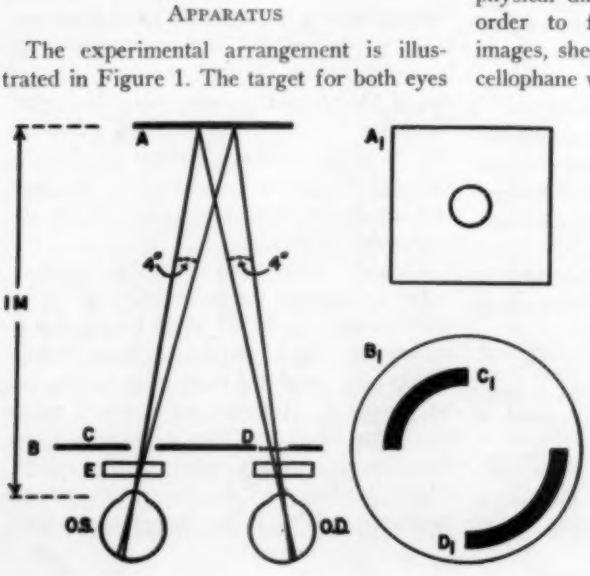


Fig. 1 (Brecher, Winters, and Townsend). Diagrammatic sketch of apparatus used for comparing right and left ocular images by image alternation. Target screen in top view (A) and in front view (A_1). The target is a circle subtending four degrees at the nodal point of each eye. Rotating disc in top view (B) and front view (B_1) has two sectors shown in top view as C and D and in front view as C_1 and D_1. Variable size lenses (E).

of the disc (yellow for one eye and green for the other), each reducing the brightness by approximately 50 percent.

Procedure

Twelve normal young subjects with undisturbed binocular vision and a visual acuity of at least 20/20 uncorrected were tested, first, with the space eikonometer (office model of the American Optical Company) and then with the method of alternating images (fig. 1).

The subject had first to become familiar with comparing size and shape of alternating images in the absence of size lenses. When the subject noticed difference in size and shape of the images, size lenses were introduced and the subject was asked to adjust the lenses until the images appeared as nearly equal as possible. Both over-all and meridional size lenses were used, the latter at axes zero degrees, 45 degrees, 90 degrees, and 135 degrees. The adjusting of the lenses for the best possible image equality was repeated by the subject five times in each of the meridians as well as for the over-all magnification. Each subject was tested in this manner on three different days. The procedure of letting the subjects adjust their own setting of the lenses for "image equalization" proved more practical than giving the subjects choices from random magnifications and distortions set by the experimenter.

Results

Upon alternating presentation to the right and left eye the objectively circular target appeared subjectively slightly noncircular to all observers. The subjective shapes and sizes of the circle varied considerably from one person to another. Several people reported "humps" or bulges in various parts of the circle.

The deviation from the objective circle was relatively constant for each eye of each observer, though changes of the subjective configurations were sometimes noticed during an experimental period and/or from one period to another. In addition, each of the images had small, fluctuating, ripplelike or scalloplike irregularities.

When the alternation of the images was suddenly stopped in such manner that after the stop the target was seen uninterruptedly by one eye only, then the distorted appearing monocular image became suddenly a "perfect circle." The same result could be achieved by covering one eye while the disc rotation continued. The process of transition of the "distorted image" to a "perfect circle" was imperceptible. Targets with other patterns such as a line, triangle, or square, appeared also slightly distorted upon image alternation.

It is obvious that any attempt to correct the above described size and shape differences of circular images can result at best in a more or less crude approximation of the

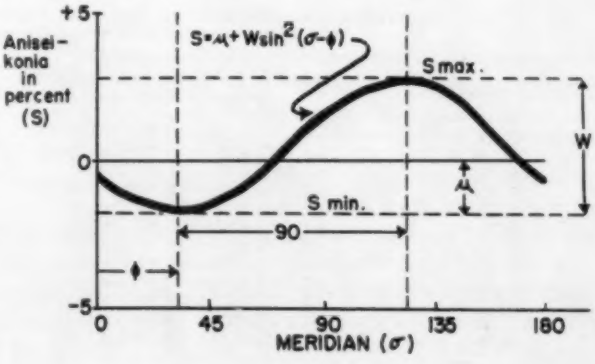


Fig. 2 (Brecher, Winters, and Townsend). Diagram of the relation of the amount of aniseikonia(s) to the meridian (σ). Horizontal broken lines indicate the maximal and minimal values of s. Vertical broken lines indicate the principal meridians. For equation and values see text.

images to circles. The available meridional size lenses permit merely correction of elliptic images but not of the slightly irregular configurations which were found to be quite common. With this consideration in mind one cannot anticipate that the subjects, who are asked to adjust the size lenses so as to produce two like circles, can reproduce the same setting with great accuracy.

An attempt was made to determine the best possible aniseikonia correction with conventional size lenses for the observed subjective irregular patterns. These corrections were based on the average values of the data which were obtained by letting the subject vary the size lenses for "image equalization."

Assuming that an aniseikonia is elliptical, then the amount of the aniseikonia will vary from meridian to meridian. The manner in which this variation occurs is illustrated by a sine function curve in Figure 2. This curve can be expressed by the equation:

$$(1)\quad s = u + w \sin^2 (\sigma - \phi)$$

where

- s = percentage of total aniseikonia in the meridian
- u = over-all aniseikonia (s min)
- w = meridional aniseikonia (s max − s min)
- σ = a meridian of aniseikonia
- ϕ = meridian of minimum aniseikonia

Although measurements in three meridians suffice for calculating an aniseikonic correction, the accuracy of the results derived from empirical data increases with the number of measurements. Since size differences were measured in four meridians with the image alternating method, the three equations for the three meridians (180, 45, 90 degrees) were supplemented by using three additional equations for including in the results the data obtained for the 135 degree meridian. The values of the meridians 45, 90, 135, and 180 degrees are substituted for σ in equation 1 in order to obtain two sets of three equations which can be solved for $\tan 2\varphi$, w and u.

for 180°, 45°, 90°

$$(2)\quad \tan 2\phi = \frac{s_{90} + s_{180} - 2s_{45}}{s_{90} - s_{180}}$$

$$(3)\quad w = \frac{s_{90} - s_{180}}{\cos 2\phi}$$

$$(4)\quad u = \frac{s_{180} + s_{90} - w}{2}$$

The calculated correction for aniseikonia is then, for the right eye, an over-all magnifier of u percent magnification, combined with a meridional magnifier of w percent magnification with the axis at ϕ.

The calculated values of the aniseikonic corrections obtained in this manner from the measurements with image alternation are presented in the left column of Table 1. The calculated values of the aniseikonic corrections obtained in the same subjects with the

TABLE 1

ANISEIKONIC CORRECTIONS CALCULATED FROM MEASUREMENTS WITH IMAGE ALTERNATION AND WITH SPACE EIKONOMETER

Subject	Right Eye Correction from Image Alteration Method	Right Eye Correction from Space Eikonometer
L. H. B.	−0.45%×38 ∞ −0.05%×128	−0.85%×16 ∞ +0.45%×106
M. A. S.	−2.40%×35 ∞ −0.70%×125	−0.90%×31 ∞ +0.20%×121
B. T.	+0.80%×41 ∞ −1.00%×131	−0.80%×19 ∞ +0.70%×109
C. C.	+0.55%×68 ∞ −0.60%×158	−0.55%×28 ∞ +0.30%×118
J. W.	+1.05%×42 ∞ −0.85%×132	−0.70%×45 ∞ +1.40%×135
E. R.	+1.05%×63 ∞ −0.50%×153	−0.30%×75 ∞ −1.70%×165
V. G.	−0.55%×14 ∞ +0.25%×104	−0.90%×62 ∞ +0.75%×152
V. P.	+0.25%×72 ∞ −0.45%×162	−1.10%×27 ∞ −0.20%×117
T. K.	+0.05%×40 ∞ −2.25%×130	−1.10%×55 ∞ 0.00%×145
M. L. K.	−2.65%×43 ∞ +3.80%×133	−1.10%×42 ∞ +0.65%×132
T. B.	+2.60%×54 ∞ −2.10%×144	−1.90%×24 ∞ +1.00%×114
B. B.	−0.15%×35 ∞ +1.60%×125	−0.40%×90 ∞ −0.75%×180

space eikonometer[8] are presented in the right column of Table 1. With eikonometry one measures only the relative differences in perceived image size and shape; consequently one can express aniseikonic corrections in reference to either eye. The corrections listed in Table 1 are given for the right eye as two meridional magnifiers with their axes crossed at right angles.

From the calculated data presented in Table 1 one may conclude that there is no consistent correlation between the corrections arrived at by the measurements with the space eikonometer and with the image alternation method.

Discussion

Alternation of the images of the right and left eye without accomplishing binocular fusion furnishes a new and simple methodologic approach for studying the relative subjective sizes and shapes of ocular images. The experiments revealed that in normal subjects each unocular image is slightly incongruent with the "object" insofar as the perceived image of a circle is only of a general circular configuration but not a perfect circle. Whereas, in current eikonometry the configuration of the perceived images can be evaluated at only a limited number of points (standard eikonometer) or can be gauged from their binocular integrating effect (space eikonometer) the method of image alternation reveals discrepancies of object and image, both uniocularly and binocularly.

With alternate presentation of the target to both eyes it was possible to distort the appearance of the target. This indicates that the distorted appearance depends upon binocular interaction. On anatomic grounds one must conclude that this occurs at the cerebral cortical level. Binocular interaction has been revealed to occur in these experiments at lower frequencies of alternation and longer durations of interruption than commonly known. Nevertheless this binocular interaction occurred without the accomplishment of binocular integration. That binocular integration was not achieved was demonstrated by the separate appearance of the images of the two eyes (lack of binocular fusion). It should be mentioned that ocular movements during the periods of alternating exposure do not account for the observed phenomenon since the same results could be obtained with much shorter exposure times (10 milliseconds).

The method of image alternation may, therefore, provide a suitable tool for studying processes of central cortical integration of binocular excitation patterns. An analysis of such processes has been published recently by Schubert.[9] The process of central integration provides a mechanism by which the target appearance resulting from disparate excitation patterns from the two eyes can be modified to resemble that produced by a simple familiar form. This integration process would serve to explain why most people tolerate unequal retinal images without awareness or complaint, for example, aniseikonias of over 10-percent size differences when viewing objects under asymmetric convergence. In fact, it makes it understandable that, within limits, slightly distorted optical images of known objects appear undistorted.

The very finding that with the image alternation method the perceived images of both eyes differ from each other in an irregular manner makes it hard for a subject to judge which aniseikonic corrections give image equality when the available meridional and over-all magnifiers are used. This difficulty may in part explain that for some sub-

for 90° 135°, 180°

$$-\tan 2\phi = \frac{s_{90} + s_{180} - 2s_{135}}{s_{90} - s_{180}} \tag{2a}$$

$$w = \frac{s_{90} - s_{180}}{\cos 2\phi} \tag{3a}$$

$$u = \frac{s_{180} - s_{90} - w}{2} \tag{4a}$$

jects there is no correlation between the aniseikonic corrections obtained with the image alternation method and with the space eikonometer (Table 1). Also the reproducibility of the results was better for the measurements with the space eikonometer than with the image alternation method. Another factor, which should be taken into account in comparing the results obtained with the two methods, is the small degree of aniseikonia existing in normal subjects. Whatever method is used in such cases, it can reveal only image discrepancies near the threshold of difference perception. A future study concerning a correlation of findings obtainable with various methods may be facilitated by the use of marked artificially induced or clinical aniseikonia.

From the foregoing it appears that the image alternation method for aniseikonia determination may be primarily of value for those persons who have poor binocular fusion and who can, therefore, not be measured with the presently available methods.

Summary

Size and shape of the perceived images of the two eyes were compared by alternating the images of the two eyes slowly enough to prevent binocular fusion but still fast enough for permitting a comparison (one to two c.p.s.). Upon alternating presentation to the right and left eye an objectively circular target appeared subjectively slightly noncircular.

Preliminary tests were made in order to find out whether image alternation could be used as a method for aniseikonia determination which might be of value for persons with poor binocular fusion. In 12 normal subjects image differences were corrected by the use of over-all and meridional size lenses.

The aniseikonic corrections obtained with the image-alternating method and with the space eikonometer did not reveal a consistent correlation. The method of image alternation appears to provide a suitable tool for studying processes of central cortical integration of binocular excitation patterns. This integration would serve to explain why most people tolerate unequal retinal images without awareness or complaint and why aniseikonia relatively seldom causes a binocular disturbance of clinical importance.

Department of Physiology,
Emory University, Georgia.

References

1. Bannon, R. E.: Space eikonometry in aniseikonia. Am. J. Optomet. Monograph 144, February, 1953.
2. Ogle, K.: Binocular Vision. Philadelphia, Saunders, 1950.
3. Bannon, R. E.: On the technique of measuring and correcting aniseikonia. Am. J. Optomet., **18**:4, 1941.
4. Brecher, G. A.: Aniseikonie und fusion. Arch. f. Ophth., **147**:17-53, 1944.
5. ———: A new method for measuring aniseikonia. Am. J. Ophth., **34**:1016-1021, 1951.
6. Hosaka, A.: Clinical studies in aniseikonia. Ochanomizu Igaku Zasshi, **3**:325-374, 1955. (Abst. by R. E. Bannon in Aniseikonic Forum, 1956.)
7. Brecher, G. A.: Quantitative studies of binocular fusion. Am. J. Ophth., **38**:134-141, 1954.
8. American Optical Company: Magnification Tables for use with the Space Eikonometer, 1948.
9. Schubert, G.: Wahrnehumungsmodulation und ihre neurophysiologischen Grundlagen. Ztschr. f. Biol., **108**:24, 1956.

SIDEROSIS BULBI AND ITS SPONTANEOUS CLEARING

A REVIEW AND CASE REPORT

MALVERN C. HOLLAND, M.D.
New Orleans, Louisiana

In the past two decades particularly, there has been a marked decrease in the incidence of siderosis bulbi. This is largely due to two factors: (1) Industrial advances, such as safety devices of various types, improved engineering, personnel training programs, and so forth, and (2) early surgical removal of iron-containing intraocular foreign bodies.

Although the ultimate prognosis in this disease is usually grave, if untreated, Duke-Elder[1] comments that there is an "extraordinary paucity in the literature of long-term observations of such cases." The patient herein reported was observed for almost 32 years and presented a rather interesting clinical course.

REVIEW OF LITERATURE

Depending on the area where the retained intraocular iron fragment comes to rest, its chemical composition, size, and other less well-known factors, gross evidence of siderosis may occur in as little time as 18 days,[2] five days[3] or only after many years, but most commonly develops in from two to six months after the injury. However, siderosis bulbi does not invariably occur in such instances for cases are recorded of iron foreign bodies being present within the globe for as long as 67 years[4] without gross reaction, though blindness often results from the initial trauma. Particles of iron as small as 0.18 mg. may be sufficient to cause siderosis.[5] Alloyed and nonmagnetic steel resist corrosion and oxidation more than do carbon steel or iron when bathed in the intraocular fluids.[6]

Siderosis is said to develop even from steel imbedded in the optic nerve[7] and in the orbit[8] but obviously there is more danger when it is within the globe, especially in the ciliary body or vitreous. In the anterior chamber the degradation products are more easily eliminated from the eye with consequently less damage. When imbedded within the lens, siderosis is somewhat less apt to occur[1] and often requires a longer period for development.

There are two main types of siderosis bulbi as described by Bunge.[9] Direct siderosis is limited to the localized pigment and cellular damage immediately about the iron-containing foreign body, whereas indirect siderosis is the diffuse involvement of subjacent or remote intraocular structures.

The exact chemical state of the iron within the siderotic eye is in dispute. Whether it is colloidal ferric hydroxide, as Mayou[10] insisted, or ferric ions in low concentration which combine with sulfhydryl groups, as considered by Friedenwald,[11] epithelial cell affinity for this toxic product is a striking feature. Wolff[12] regarded this affinity only as an indication of the high metabolic rate of these cells.

Retinal ganglion cells after receiving these deposits degenerate and become replaced by glial cells; retinal pigment epithelium takes up the iron and invades the inner retinal layers to collect about blood vessels, resulting in local vascular sclerosis and, thence, further retinal atrophy.[10]

Nyctalopia, peripheral contraction of the visual fields followed often by optic atrophy, retinal detachment, and, later, phthisis bulbi represent the clinical sequence commonly encountered.[1]

The iris muscles,[13] epithelium of the lens, and, according to Loewenstein and Foster,[14] Bowman's membrane as well as the zonule are densely infiltrated with iron compounds. Other "glass membranes" and occasionally the macular area may relatively escape. Preferential staining of the iron is shown by the

Perl's Prussian blue reaction utilizing potassium ferrocyanide and hydrochloric acid. Hemorrhage within the eye may complicate the histochemical picture because hemosiderin liberated from old erythrocyte accumulations also gives a positive Perl's reaction. However, hemoglobin not acted upon by cells of the reticuloendothelial system gives a negative reaction.[15]

Mydriasis of the brown-hued iris often occurs and the cataractous lens with its brittle zonule may subluxate,[16] or dislocate[14] after little or no known trauma. Early in the disease the lens epithelium[17] proliferates and concentrates intracellularly the brownish-red pigment so characteristic on biomicroscopic examination even in the absence of traumatic capsular damage.

The injured eye containing iron may never reach the full-blown siderotic state because of the rather common occurrence of iridocyclitis and secondary glaucoma which may develop early or late after foreign body injury even without siderosis[18] and necessitate enucleation. Glaucoma associated with siderosis offers a poor prognosis[20, 21] and responds poorly to fistulizing operations.[1]

Contrary to d'Amico's point of view[22] it is now generally agreed that up to a limit the iron-tissue combination is reversible with preservation of cell function providing the source of the diffusible iron is removed.[23, 24] Perhaps the electroretinographic findings of early increased positive and decreased negative potentials noted by Karpe[25] in early siderosis represent the stage during which the major part of the retinal function will return if the iron foreign body is extracted from the globe. Later the electroretinogram becomes negative or shows no response.

Many reports are in the literature of disappearance or reduction of brown discoloration of the iris and lens after surgical removal of the foreign body, including those of Vossius,[16] deSchweinitz,[23] Sweet,[26] Black,[27] and Cridland.[18] Mydriasis may disappear and iris reflexes return as soon as one month after removal.[26]

There have been several reports of good vision following indirect siderosis of more than a few years' duration. Flynn and Raiford[28] reported a case of magnetic foreign body in which only slight siderosis of the anterior segment occurred after 24 years. Removal of the cataractous lens containing the particle resulted in good vision. Begle[29] reported two cases in which such particles appeared to have fully "rusted," becoming nonmagnetic as tested when removed eight years and one year, respectively, after injury.

Spontaneous resorption

The cases reported wherein spontaneous clearing of siderotic pigment without surgical intervention are indeed rare. Von Hippel,[30] in an exhaustive report, correlated to some degree the decreasing amount of pigment in many siderotic eyes with the increasing interval between injury and enucleation. However, he concluded that since old hematogenous pigment could not be distinguished from exogenous iron in the tissues, definite absorption of the latter was not established. Vossius[31] and Mayou[10] felt that a very small iron foreign body could spontaneously absorb but offered no clinical material to substantiate this belief. Hirshberg,[32] in writing of the first ocular use of the electromagnet, states that foreign bodies five to 10 mg. in weight may absorb, but others have considered this estimate overly optimistic. Cridland[18] recorded a case of about seven years' duration without surgery in which iris discoloration disappeared but the eye had become blind.

Braendstrup[33] reported two cases of spontaneous absorption involving minute ferrous intraocular foreign bodies. In one case the exogenous pigment had grossly cleared in six years although evidence of this pigment persisted on biomicroscopic examination. Radiographic evidence of the foreign body had disappeared without surgery. There was iris and choroidal atrophy without restricted visual field and vision was about 20/400

with moderately advanced cataract. The other case, probably of about two years' duration prior to discission of cataract, had corrected vision of 20/20 with aphakic lens and "no trace of siderosis at all" when next seen 14 years later. No field examination was mentioned. In Goulden's case[24] the foreign body disappeared to X-ray examination in "several" years although gross iris discoloration persisted.

Comment

It is, I think, important to emphasize especially three points in evaluating any case report on siderosis bulbi in which there are positive followed by negative radiographic studies. First, it is not rare to have an iron-containing intraocular foreign body of size adequate to cause siderosis yet too small to be visualized by X rays. Especially is this true regarding older reports when only large focal spot X-ray tubes and poor film emulsions were available. Rychener's case[25] illustrates such difficulty even with modern techniques. An iron foreign body of one year's duration causing indirect siderosis could be seen within the lens but could not be demonstrated by X ray. The foreign body was found to be still weakly magnetic when obtained at the time of linear cataract extraction. Davidson[9] found three of 15 relatively early cases to have negative X rays.

Secondly, in a nonoperated siderotic eye that shows no radiographic evidence of a previously demonstrated foreign body, one of two possibilities exists. The particle may have been reduced in size below the minimum demonstrable by X ray yet remain as a magnetic corroding object or it may have completely disintegrated into a mass of relatively radiolucent, nonmagnetic, siderotic pigment. In either instance a potentially dangerous and destructive situation for the eye exists even when encapsulation has occurred.

The third possibility which becomes apparent only after many years' observation is that the foreign body is completely absorbed or reaches an equilibrium state wherein further damage to the eye is not apparent although, undoubtedly, some iron pigment could be detected in the pathologic specimen. Into this relatively rare group the following case report belongs.

Case report

Clinical history. In December, 1924, this white seaman, now aged 62 years, received a perforating steel foreign body injury to the left eye. The steel was thought to be from a hatchet used as a wedge which the patient was hitting with a hammer at the time of injury. A small superficial corneal foreign body was removed from the corneal surface of the eye by the patient's family doctor on the same day. Following two weeks of topical therapy the eye became essentially asymptomatic except for slightly decreased vision and occasional mild redness without pain, not requiring treatment.

About two months later this normally blue-eyed man noted his injured eye to be turning brown in the iris area but sought no medical care until December 12, 1925, one year after injury.* At that time vision was: R.E., 20/15; L.E., 20/40. There was mild conjunctival injection, a small through-and-through corneal scar, brownish discolored iris with iridoplegia, and the pupil measured eight mm. Fine bricklike dust was on the anterior lens surface in blotches and posterior capsular and subcapsular opacities obscured fundus details. A diagnosis of siderosis bulbi was made. Localization films revealed a midline 0.5 mm. in diameter radiopaque foreign body eight mm. posterior to the corneal center and nine mm. below the horizontal plane. The giant magnet gave no observable pull on this particle. Of four ophthalmologists seeing this patient, two recommended surgical removal of the foreign body but the patient declined.

When next seen 29 months after injury, vision in the left eye was reduced to finger counting at 10 feet because of increased lens opacity. However, the pupil reacted and was of the same diameter as that of the fellow eye. The iris remained brown.

On December 12, 1933, the patient's next visit, cataract surgery was discussed but the patient refused. Exactly one year later (nine years after injury) the lens was found to be spontaneously subluxated posteriorly to remain attached to its zonule only at the 6-o'clock meridian. Intraocular pressure was normal.

When next seen in December, 1936, (12 years after injury), the lens was dislocated and out of view. Vision of this eye then with pinhole disc and +9.0D. sph. was 20/50. Some ciliary congestion was evident and the patient was begun on topical

* All clinical information up to January 19, 1937, is from the records of the late Dr. Will Otto Bell, courtesy of Dr. Laurel R. Foxworthy, Medical and Dental Building, Seattle, Washington.

cycloplegic and heat. Tension was not recorded on that date but two days later was 50 mm. Hg (Schiøtz) with corneal bedewing. Redness persisted for 10 days only. Tensions varied between 45 and 62 mm. Hg (Schiøtz) but remained elevated when this patient was last seen by Dr. Bell on January 19, 1937.

The patient states that he has received no therapy for the left eye since 1937. The color of the iris subjectively remained dark brown only about two years after onset and has improved steadily since that time in comparison with the normal right iris. The eye has been without redness, pain, or other symptoms since the recorded glaucoma episode. There has been no diplopia and no irritation of the opposite eye.

Current examination. Vision was: R.E., 20/25; L.E., counting fingers at eight feet. There was left exotropia of 20 prism diopters for distance in the primary position with some intermittent suppression of the involved eye. Refraction: O.D., + 1.25D. sph. ⊃ −0.5D. cyl. ax. 40° = 20/15; O.S., +10.5D. sph. ⊃ −1.75D. cyl. ax. 105° = 20/20. Vertex distance, 11 mm. Tension (Schiøtz): O.D., 20 mm. Hg; O.S., 23 mm. Hg.

Externally, the left eye appeared essentially normal but iridodonesis was present. In moderate, diffuse lighting the right pupil measured 2.5 mm. and the left, about 3.0 mm. Both responded promptly to direct and consensual light as well as on convergence. Grossly, the left iris was slightly darker blue than the blue-gray right iris. Transillumination of the left globe revealed a small iris hole at the 3-o'clock position near its base with a somewhat deepened anterior chamber. The lens was absent from the pupillary area.

Ophthalmoscopically, the optic nervehead and retina were most interesting. The optic disc was pale with diminished number of surface capillaries, especially in the lower temporal quadrant, but showed no evidence of glaucomatous cupping. Diffusely and focally throughout the retina pigmentary degeneration and migration were prominent, giving a generalized mottled and tessellated appearance not present in the right eye. This patchy pigmentation blended rather evenly into zones of relatively normal-appearing retina. Some prominence of this pigment about the retinal vessels was evident and in other areas there was less than normal pigment in the retina, thus allowing visibility of choroidal vessels.

The macular area was generally hyperpigmented but had none of the clumps noted elsewhere in the retina.

The second striking retinal finding was located inferiorly and temporally. This consisted of several large whitish to ivory-colored, sharply circumscribed, atrophic area of healed chorioretinitis. At least one consultant considered an area which was whiter than the others situated at the 6-o'clock position in the equatorial region to represent the capsular residual of the dislocated lens.

The third, and perhaps the most physiologically significant ophthalmoscopic finding, was of marked attenuation of all the retinal vessels—principally of the arteries and arterioles—quite out of proportion to the minification expected in aphakia. Increased vein/artery ratio existed. In a few areas, especially after the second bifurcation of the inferior retinal arteries, almost complete obliteration of the red reflex was observed in segments of these sclerotic arterioles.

The patient's blood pressure was 186/82 mm. Hg, and the right fundus revealed only minimal Grade I arteriosclerotic retinopathy.

Biomicroscopic examination revealed a normal right eye.

The left cornea at the temporal limbus had a through-and-through scar overlying the iris hole. Otherwise, the cornea was clear, of normal thickness without discernible pigmentation in any layer. By specular reflection the endothelium was intact.

The iris, with its delicate structure, gave only minimal suggestion of atrophy. Its surface, excluding the crypts, was apparently free of exogenous pigment. However, the crypts had a peculiar type of evenly distributed granular, particulate, brownish-red pigment deposited on the surface of the crypts as well as within the iris stroma in these areas. Although it could not be definitely distinguished in appearance from uveal pigment, it seemed of redder color. These deposits were predominantly in the midzone of the iris outside the collarette and decreased as one approached the base. No keratic precipitates or synechias were present and the anterior chamber was clear.

A relatively flat vitreous hyaloid membrane was present without enmeshed pigment cells, discoloration, opacities, or herniation.

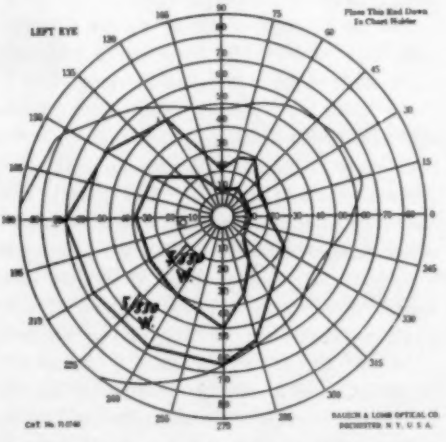


Fig. 1 (Holland). Peripheral field, using five-mm. and three-mm. white test objects at one-third meter.

Central and peripheral fields were normal in the right eye but definite changes in the left eye were found. Using the Feree-Rand perimeter with seven foot-candle illumination, a general contraction with pronounced deficit in the superior and nasal field was found (fig. 1). Central fields (fig. 2) with 1.0 mm., 3.0 mm., and 10-mm. white Berens' test objects again showed marked contraction, especially in the superior and lower nasal quadrants. Each of these examinations was performed with aphakic correction in the trial frame with appropriate add for the working distance, that is, plus 3.0D. for the perimeter and plus 1.0D. for the tangent screen. Reduced illumination caused a marked increase in the peripheral contraction of the visual field.

Stereoscopic face-down views with central ray directed 20 degrees toward the feet showed no evidence of metallic foreign body within the orbits. Two lateral views using the nonscreen technique were taken and again there was no evidence of radiopaque foreign body.

Discussion

This is a unique case of siderosis bulbi in which remarkable absorption of a small iron-containing intraocular foreign body has occurred with preservation of excellent corrected vision. Progressive subjective and objective clearing of the pigmented iris has taken place spontaneously. Some exogenous pigment persists and is best seen with the biomicroscope in the crypts of the iris where normally the endothelium and anterior limiting membrane are anatomically absent. Undoubtedly, this pigment exists in other areas of the iris but is obscured by the above layers where they are present.

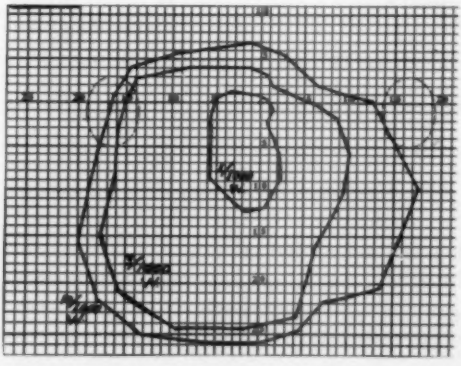


Fig. 2 (Holland). Central field, using one-mm., three-mm., and 10-mm. test objects and one-meter tangent screen.

Mydriasis so often reported to be in association with siderosis was transiently present and return of reflexes roughly coincided with the patient's observation of lessened iris pigmentation.

Extensive effect of old retinal disease is prominent and, in conjunction with the inherent peripheral distortion produced by the aphakic lens, accounts for the field defects and perhaps for the occurrence of retinal fatigue noted during field examinations. In the presence of such an excellent corrected visual acuity it is obvious that the macular area has escaped most of the devastating effect of siderotic change as did the case of Loewenstein and Foster.[14] They found no histochemical evidence of macular iron in an eye with siderosis bulbi for 22 years.

The patient's exotropia and lack of interest, in addition to recently diagnosed carcinoma of the stomach, negated the possibility of a successful contact lens fitting for the involved eye. Hence, this step toward binocular single vision was not attempted.

The brief glaucoma episode was not typical of the type usually seen associated with siderosis bulbi and appeared to be directly related to the cycloplegic used. A chamber angle already embarrassed by siderotic pigment and dislocated lens was thus further compromised by a dilated iris. The elevated intraocular pressure cleared when iris reflexes returned. At the current examination there was no clinical evidence of past or present uveitis or glaucoma.

Some degree of conservatism in the handling of intraocular iron-containing foreign bodies has been recommended by Braendstrup,[33] Bulson,[19] Clegg,[20] and others. Rollet,[36] reporting on 222 cases, noted that such foreign bodies less than 0.2 mg. in size could not be effectively removed with the giant magnet. Each patient, of course, has to be considered individually and rarely is surgical removal of the foreign body not attempted.

In the case of a very small posterior segment foreign body where reasonable and judicious efforts at removal have been made, utilizing all the modern techniques at hand without success, one should not be overly discouraged. Such cases do not always lead to blindness. *Primum non nocere*—"First do no harm."

Summary

A case of siderosis bulbi of almost 32 years' duration is presented. In the course of his disease this patient developed brown iris pigmentation which grossly cleared, cataract with subsequent dislocation, transient mydriasis, and a brief episode of secondary glaucoma. Without surgical intervention the previously demonstrated intraocular foreign body of 0.5 mm. diameter is now radiologically and clinically absent. His central visual acuity with aphakic correction is 20/20. Present findings due to siderosis include aphakia, microscopic iris pigmentation, retinal atrophy with vascular attenuation, chorioretinal scarring, field defects, and minimal optic disc pallor.

210 State Street (18).

References

1. Duke-Elder, S.: Textbook of Ophthalmology. London, Kimpton, 1954, v. 6, pp. 6198-6214 and 6303-6308.
2. Davidson, N.: Siderosis bulbi. Am. J. Ophth., **16**:331-335, 1933.
3. Smith, H. G.: Siderosis. Am. J. Ophth., **12**:409, 1929.
4. Spratt, C. N.: Foreign body in the eye 67 years. Arch. Ophth., **9**:102-103, 1933.
5. Hüttemann, R.: Ueber die Wöhrend der Letzten 3 Jahre in der Strassburger Universitats-Augenklinik Boebachteten Eisensplitter Verletzungen der Auges. Klin. Monatsbl. f. Augenh., **51**:351-331; 473-493, 1913.
6. Ballantyne, J. F.: Siderosis bulbi. Brit. J. Ophth., **38**:727-733, 1954.
7. Santoro, N.: Siderosis bulbi: Diffuse precocious siderosis caused by metal splinter embedded in optic nerve case. Gior. ital. oftal., **2**:384-389, 1949.
8. Sironi, L.: Siderotic cataract due to metallic splinter adhering to lateral wall of orbit without penetrating eyeball. Boll. Ocul., **21**:585-593, 1942.
9. Bunge, P.: Ueber die Siderosis Bulbi. Verhandl. Internat. Med. Kong. (Berlin), **4**:151-154, 1890.
10. Mayou, M. S.: Siderosis. Tr. Ophth. Soc. U. Kingdom, **46**:167-180, 1926.
11. Friedenwald, J. S.: Cited by Ballantyne.[4]
12. Wolff, E.: The spread of iron compound in siderosis bulbi. Proc. Roy. Soc. Med., **27**:691-694, 1934.
13. Verhoeff, F. H.: Siderosis bulbi. Brit. J. Ophth., **2**:571-572, 1918.
14. Loewenstein, A., and Foster, J.: A contribution to the knowledge of ocular siderosis and posterior degenerative pannus. Am. J. Ophth., **30**:275-288, 1947.
15. Wolff, E.: A Pathology of the Eye, New York, Blakiston, 1951, p. 219.
16. Vossius, A.: Siderosis der Iris and Linse infolge intraokularen Eisensplitter. Deutche Med. Wchnschr., **36**:1108, 1910.
17. Leber, T.: Ueber die Wirkung von Fremkorpern im Innerven der Auges. Tr. Internat. Med. Cong., London, **3**:15-19, 1881.
18. Cridland, A. B.: Investigations on the "aftermath" of cases of intraocular foreign body. Tr. Ophth. Soc. U. Kingdom, **53**:438-465, 1933.
19. Bulson, A. E., Jr.: Tolerance to foreign bodies within the posterior segment of the eye. J.A.M.A., **87**:2071-2078, 1926.
20. Clegg, J. G.: Siderosis of the eye. Seven cases. Ophthalmoscope, **13**:501-507, 1915.
21. Unger, L.: Secondary glaucoma after retained intraocular foreign body. Klin. Monatsbl. f. Augenh., **126**:451-460, 1955.
22. d'Amico, D.: Siderosis of the globe. Ann. ottal. & clin. ocul., **53**:289-398, 1925.
23. de Schweinitz, G. E.: Concerning the disappearance of the coloration in siderosis bulbi. Ophth. Rec., **18**:155-156, 1909.
24. Levine, J.: Siderosis bulbi (Xenogenous). Arch. Ophth., **11**:625-634, 1934.
25. Karpe, G.: Early diagnosis of siderosis bulbi by use of electroretinography. Docum. Ophth., **2**:277-298, 1948.
26. Sweet, W. M.: Siderosis with dilated inactive pupil. Am. J. Ophth., **7**:871-872, 1924.
27. Black, N. M.: Siderosis bulbi with dilated inactive pupil. Recovery. Am. J. Ophth., **6**:990-995, 1923.
28. Flynn, G., and Raiford, M.: Magnetic foreign body: Report of intraocular case in which removal was successful after 24 years. Am. J. Ophth., **38**:90-91, 1954.

29. Begle, H. E.: Perforating injuries of the eye by small steel fragments. Am. J. Ophth., **12**:970-977, 1929.
30. von Hippel, E.: Ueber Siderosis und die Beziehunger zwischen siderotischer und hematogener Pigmentierung. Arch. f. Ophth., **40**:123-279, 1884.
31. Vossius, A.: Ein Fall von minimalem Eisensplitter in der Linse nebst Bremerkungen über die Diagnose intraocularer Eisensplitter. Deutche Med. Wchnschr., **35**:21-22, 1909.
32. Hirshberg, J.: Ueber die Ergebnisse der Magnetoperation in der Augenheilkunde. Arch. f. Ophth., **36**:37-98, 1890.
33. Braendstrup, P.: Two cases of temporary siderosis bulbi with spontaneous absorption without impairment of function. Acta. Ophth., **22**:311-136, 1944.
34. Goulden, C.: Siderosis. Am. J. Ophth., **15**:1171-1172, 1932.
35. Rychener, R. O.: Siderosis following intralental foreign body. Am. J. Ophth., **29**:346-347, 1946.
36. Rollet, E.: Small magnetic intraocular fragments removed by electromagnet. *Soc. d'ophtal.* Nov., 1921. Cited in The Ophthalmic Yearbook, **20**:360, 1924.

FUNDUS CHANGES IN INCONTINENTIA PIGMENTI*

(Bloch-Sulzberger syndrome)

Wolfgang A. Lieb, M.D., and DuPont Guerry, III, M.D.
Richmond, Virginia

A clinical entity, characterized chiefly by ectodermal defects, was described by Bloch in 1926 and given the name incontinentia pigmenti. In 1928, Sulzberger reported on the same case and, together with others, suggested in 1938 that this was part of a syndrome of other familial ectodermal defects. Changes in the skin have been more often described, but defects in the eyes, hair, nails, teeth, and central nervous system have been observed.

It is interesting to note that the case demonstrated in 1925 by Bloch to the Swiss Dermatological Society, that of a girl, aged one and one-half years, was first admitted to the eye clinic with a diagnosis of a glioma with secondary retinal detachment. She was referred to Blochs' dermatology clinic for the differential diagnosis of a peculiar skin pigmentation, consisting of bizarre, irregular spots over her trunk and right lower extremity. Such a condition had not previously been described in any dermatosis. The pigmentation was chocolate brown in color and had been present from birth. The patient was also subject to a recurring exudative inflammatory dermatitis, which, upon healing, left similar pigmented lesions.

Histologically, the changes were confined to the basal layer, where small cells with piknotic nuclei, various degrees of irregularity, and hydropic changes of individual cells were noted. The hyperpigmentation was apparently due to chromatophores containing large amounts of fine, powdery granules of melanin situated within the upper and middle cutis.

Although this clinical entity is attributed to the work of Bloch and Sulzberger, the first reference was probably made by Garrod, in 1906, who observed a "peculiar pigmentation of the skin in an infant." Adamson, in 1907, reported on similar cases.

Franceschetti and Jadassohn, in 1954, made a thorough review of the literature and collected 73 cases of the Bloch-Sulzberger syndrome to which Scott, et al., in 1955, added the review on 19 more. In 1938, Sulzberger and his co-workers reported the case of a 19-year-old girl with incontinentia pigmenti who, in addition, showed changes of the lens and a calcareous opacity of the cornea together with convergent strabismus. Other observers have noted ocular malfor-

* From the Department of Ophthalmology and the Titmus Eye Foundation of the Medical College of Virgina.

TABLE 1

OCULAR CHANGES IN INCONTINENTIA PIGMENTI
(BLOCH-SULZBERGER SYNDROME)

Ocular Changes	Observer	Cases Reported
Strabismus	Lechleutner-Siemens, 1929; Pierini, et al., 1945; Heilesen, 1948; Camey, 1951; Higuchi, et al., 1952; Lieb and Guerry, 1957	5
Nystagmus	Haber, 1952	1
Blue scleras	Sulzberger and Bloom, 1948; Haber, 1952	2
Myopia	Lovemann, Fliegelmann, Weidmann, and Rein, 1952; Rein and Weidmann, 1952	1
Cataract	Sulzberger, 1938; Jaramillo, Mantesota, and Rosselot, 1948; Curth, 1949; Gasteiger, 1951; Ito, 1951	5
Papillitis	Kühling, 1949	1
Exudative chorioretinitis	Lieb and Guerry, 1956	1
Retrolental fibroplasia	Uebel, 1950; Findlay, 1952; Watanabe, 1954	3
Pseudoglioma	Bloch-Sulzberger-Franceschetti and Jadassohn, 1954; Haxthausen, 1945; Duke University, 1948	3
Optic atrophy	Moncorps-Seidlmayer, 1942; Seidlmayer-Höra, 1943; Gerard-Lapiere, 1951; and Carney, 1951	4
Metastatic ophthalmitis	Kawamura, 1954; Scott, Friedmann, Chitters, and Pepler, 1955	2

mations in conjunction with this syndrome.

In reviewing the literature, we found that one of our cases differs in its ocular manifestations from others reported previously. Table 1 illustrates the primary ocular defects and their statistical occurrence in the literature.

CASE REPORT

A four-week-old Negro infant was admitted to the ward pediatric service for evaluation and diagnosis of a pigmented vesicular rash present since birth and believed to be incontinentia pigmenti by the dermatologists. The infant was delivered at home by a midwife after an uneventful eight-month gestation of a 23-year-old gravida V para IV abortiva O mother. The child cried and sucked spontaneously and had no difficulties with respiration.

At birth, the child was noticed to have tiny pinhead-sized vesicles on all extremities with a few similar scattered vesicular eruptions over the trunk. She was taken to her family physician, who administered penicillin intramuscularly without noticeable improvement in the lesions. At the sites of healing, deeply pigmented spots were left. The child was then hospitalized in another hospital, where several serologic tests for syphilis were run but were all reported as negative. She was then referred to our dermatologic clinic, where the diagnosis incontinentia pigmenti was made and from here to the pediatric service for confirmation of diagnosis.

Physical examination on admission showed a temperature of 99.6°F. rectally, pulse 140, respiration 50. She was a well-developed, well-nourished infant in no acute distress. The physical examination was completely negative except for the remarkable pigmentation of the skin and the eye lesions. Multiple small areas of hyperpigmentation were scattered over all extremities and, to a lesser degree, over the trunk. In some places, the pigmented areas were so thick as to be confluent, this being generally true over the extremities. Numerous small vesicles in several stages (fresh, drying, and healing) were scattered over this same pigmented area. Both lesions showed a predominantly linear distribution (figs. 1, 2, and 3).

Examination of the eyes revealed the external eye to be essentially normal.

Right fundus. The disc showed normal coloration and sharp borders. At the temporal side, there was a small pigment sickle. The superior temporal vein was engorged and showed multiple divisions resembling a pannuslike vascularization. The inferior temporal vein was equally markedly engorged. Temporal to the macular area, one branch of the inferior temporal vein was markedly tortuous and engorged, resembling a corkscrew in configuration. The whole temporal fundus, limited

Fig. 1 (Lieb and Guerry). Distribution of lesions in incontinentia pigmenti.

by superior and inferior temporal veins, showed marked retinal edema which was most distinct in the area of neovascularization. The macular area showed extensive pigment changes and the foveola was covered by a kidney-shaped, whitish degeneration and exudate. In the surrounding area, there were several star-shaped, whitish exudates. Above the macula in the area of neovascularization, there was a large elevated focus of whitish-yellow color. While the lower border of this area of localized exudation was sharply delimited, the other border was indifferent and diminished into hazy edema. In the edematous area all the venules showed increased tortuosity with "corkscrew" changes (fig. 4).

Left fundus. Optic disc and retinal vessels, with the exception of the macular region, appeared normal. In the macular area, there was extensive edema and corkscrew vessels. No foveal reflex was observed. The arterioles showed no changes.

Admission laboratory work showed a hemoglobin of 10.2 gm. percent; white count 11,900 with 30-percent polymorphonucleocytes, 67-percent lymphocytes, one-percent eosinophils, one-percent basophils, and one-percent monocytes. Urinalysis was negative except for a one-plus albumen urea. Sickle cell preparation was negative. Blood cultures showed no growth. Films of the chest and long bones were not remarkable. Electrocardiogram revealed only a sinus tachycardia. Skin biopsies from areas of pigmentation showed the following histologic findings:

"A moderately acute inflammation of the epidermis and upper dermis. The epithelium in a large segment shows moderate acanthosis and prolongated rete pegs. A characteristic feature is the presence of dyskeratosis associated with melanin pigment. Considerable pigment in melanophores is seen in the dermis. There is also a perivascular infiltrate of lymphocytes in the upper dermis. Pathologic diagnosis: incontinentia pigmenti of leg and forearm."

The patient's hospital stay was uneventful except for one temperature elevation, due to an upper respiratory infection which responded rapidly to antibiotics. She was discharged after one week's hospitalization to be followed jointly in the pediatric and dermatology out-patient clinics.

Fig. 2 (Lieb and Guerry). Linear distribution of lesions on the trunk.

Fig. 3 (Lieb and Guerry). Confluency of lesions on lower extremities.

Discussion

In 1954, after a thorough review of the then current literature, Franceschetti and Jadassohn made a distinction between two groups of "incontinentia pigmenti." The first, or Bloch-Sulzberger type, occurs almost exclusively in females and is distinguished by a splashlike pigmentation and absence of malformations. The second, or Naegeli type, is found in both sexes and is characterized by the presence of a reticular-type pigmentation in association with various malformations.

There is strong evidence to support the

Fig. 4 (Lieb and Guerry). Exudative chorioretinitis with neovascularization in incontinentia pigmenti (Bloch-Sulzberger syndrome).

theory that this is an inherited or congenital defect. Cochayne feels that the presence of the condition in uniovular twins as reported by Naegeli is good evidence that it is inborn. The fact that it was present in a father and his two children indicates that it may be inherited as a dominant characteristic. Curth's cases, and those of Philpott, et al., follow the pattern of dominant inheritance. A familial pattern is also reported by Naegeli, Sobel, Levin, Asboe-Hansen, and Kitamura, et al.

It has been suggested that incontinentia pigmenti may be the end-result of a viral infection of the mother during pregnancy. In 1950, Miller, et al., reviewed the literature with respect to congenital malformations due to dermatotrophic virus infections in gravid women. Besides rubella, the viruses of Herpes zoster, scarlatina, varicella, smallpox, epidemic parotitis, infectious hepatitis, and poliomyelitis, may cause developmental fetal anomalies such as cataracts, glaucoma, microphthalmia, mental defects, cardiac and dental abnormalities following infection of the mother during pregnancy. Harber also

emphasized the similarity in ectodermal and mesodermal defects of incontinentia pigmenti and those which result from infections with dermatotropic viruses in pregnant women. In 1954, Murrell and Lingamfelter had a case which, from the histopathologic standpoint, presented a picture compatible with a viral infection. A biopsy from a hyperkeratotic lesion showed a verrucous papilloma with a number of basophilic dyskeratoses somewhat resembling molluscum bodies, confined to the stratum comeum. The molluscumlike dyskeratosis resembled those of the Shope tumor of rabbits, suggesting the possibility of a viral etiology.

The first clinical reports were limited to descriptions of the pigmentary phase and the associated developmental anomalies. Carney, in 1951, was the first to stress the initial vesicular stage which often precedes the better known, bizarre pigmentary

←

Fig. 5 (Lieb and Guerry). Skin changes in incontinentia pigmenti: dyskeratosis, acanthosis, and enlargement of the rete peg. Increase in clear cells and hyperpigmentation in the epidermis.

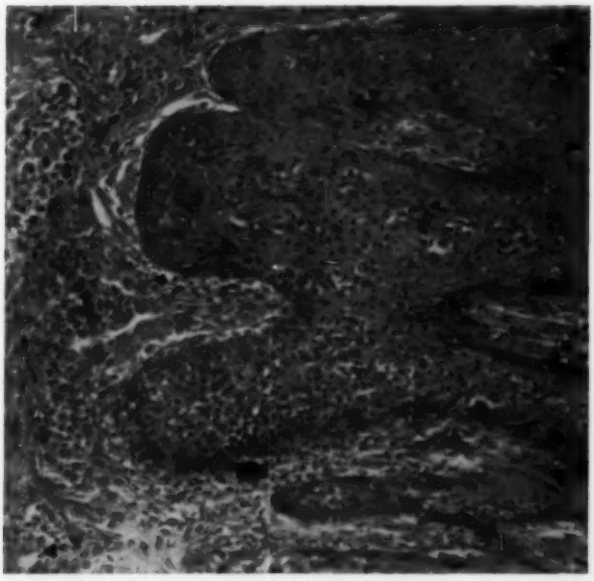


Fig. 6 (Lieb and Guerry). Incontinentia pigmenti: increase in clear cells in the epidermis with clusters of melanophores in the upper dermis.

changes. During the initial stages, recurrent crops of inflammatory lesions, primarily vesicles and bullae, may appear in association with blood eosinophilia. Later, linear verrucous lesions develop which, in the end-stages, give way to the typical pigmentation by which this disease is characterized.

Various ectodermal and mesodermal defects accompany this disorder. Findlay found associated eye disorders in 25 percent of the cases. In our case, the dermatologic picture was characterized by vesiculopapular eruptions distributed in linear fashion, irrespective of nerve distribution over the lower extremities, vulvae, arms, thighs, and trunk with greatest concentration over the extremities. Many of the vesicles demonstrated an increase of pigmentation at the base. A few of the lesions showed a verrucous surface.

Histologically, the skin biopsy specimen presented an inflammatory infiltrate in the epidermis and upper dermis with a hyperkeratosis and acanthosis. There was a perivascular infiltrate of moderate degree in the upper cutis. From the clinical picture and histologic sections, the diagnosis of incontinentia pigmenti was substantiated.

The eye disorders were restricted to the fundi. The changes consisted of marked retinal edema, primarily between the superior and inferior temporal vessels and extending from the temporal disc margin to the periphery. There was extensive engorgement of these vessels and some of the smaller venous branches showed marked tortuosity. Aberrant vascularization was a distinct feature, especially in the superior temporal area. In the macula, a kidney-shaped fatty degeneration covered the fovea. Surrounding this, there was a star-shaped area of degeneration and exudation. Focal areas of exudation with elevation completed the picture. This whole lesion resembled an exudative type of chorioretinitis. If there is a relation to cases in the literature, these changes appear to be most closely related to those eight cases collected by Scott, which he called "a mass in the orbit." Of this group, two resembled metastatic ophthalmia; three, retrolental fibroplasia; and three, a pseudoglioma. As to the etiology of the fundus pathology in our cases, not having the advantage of histologic sections, we can only speculate on the pathogenesis.

The recent work of Wise, demonstrating fundus pathology after venous occlusion from any cause, suggests the possibility that some such process might be operative here, particularly since the fundus picture somewhat resembles that of an early angiomatosis or even Coats' disease. Because the lesion was present bilaterally, though to a lesser extent on one side, we favor a congenital defect. However, we cannot exclude a viral infection because, had such an infection occurred intrauterine, it is highly possible that the lesion could still be bilateral in occurrence as, namely, the congenital cataracts following a rubella infection of the mother during gestation.

Despite the questionable etiology, whether hereditary or original, we might be able to consider our ocular findings as an early or rudimental stage of a disorder which may later be found in the end-stages to be resembling metastatic endophthalmitis, retrolental fibroplasia, or pseudoglioma.

Certainly, as the problem is better understood, a revision of nomenclature will become a necessity. This fact has already been mentioned by Ashton in 1954 and Scott, et al., in 1955.

Summary

1. A case of exudative chorioretinitis occurring in conjunction with incontinentia pigmenti (Bloch-Sulzerger syndrome) in a Negro infant is presented with illustrations of the skin and fundus changes.

2. A statistical analysis of the ocular defects accompanying this disorder has been done.

3. Various theories of the etiology are discussed.

Medical College of Virginia (19).

References

Adamson, H. G.: A circinate eruption diagnosed in India as "Dhobie itch." Brit. J. Derm., **19**:199, 1907.

Asboe-Hansen, G.: Bullous keratogenous and pigmentary dermatitis with blood eosinophilia in newborn girls. Arch. Derm. & Syph., **67**:152, 1953.

Ashton, N.: Pathological basis of retrolental fibroplasia. Brit. J. Ophth., **38**:385, 1954.

Bloch, B.: Eigentümliche bisher nicht beschriebene Pigmentaffection. Schweiz. med. Wschr., **7**:404, 1926.

Carney, R. G.: Incontinentia pigmenti: A report of five cases and a review of the literature. Arch. Derm. & Syph., **64**:126, 1951.

Cochayne, A. E.: Inherited abnormalities of the skin and its appendages. London, Oxford, 1933, p. 325.

Curth, H. O.: Follicular atrophoderma and pseudopelade associated with chondrodystrophia calcificans congenita. J. Invest. Derm., **13**:233, 1949.

———: Incontinentia pigmenti in a girl aged 13 months. Arch. Derm. & Syph., **60**:970, 1949.

Duke University: Case report. Baltimore-Washington Derm. Soc., **13**:11, 1948.

Findlay, G. H.: On the pathogenesis of incontinentia pigmenti: With observations on an associated eye disturbance resembling retrolental fibroplasia. Brit. J. Derm., **64**:141, 1952.

Franceschetti, A., and Jadassohn, W.: A propos de l'incontinentia pigmenti, delimitation de deux syndromes différents figurant sous le même terme. Dermatologica, **108**:1, 1954.

Garrod, A. E.: Peculiar pigmentation of the skin of an infant. Tr. Clin. Soc. Lond., **39**:216, 1906.

Gasteiger, H.: Incontinentia pigmenti. Ber. Deutsch. Ophth. Ges., **57**:292, 1951.

Haber, H.: The Bloch-Sulzberger syndrome (incontinentia pigmenti). Brit. J. Derm., **64**:129, 1952.

Haxthausen, H.: Incontinentia pigmenti. Acta derm.-venereol. (Stockh.), **25**:528, 1945.

Heilesen, B. J.: Incontinentia pigmenti. Acta derm.-venereol. (Stockh.), **28**:544, 1948.

Higuchi, K., Urabe, H.: Hirohaka, T., and Hirata, T.: Cited by Kitamura: Hihu-to-Hitsuhyo, **14**:109, 1952.

Höra, J.: Nebennieren markaplasie und eigenartige Hautpigmentanomalie. Zentralbl. allg. Path. path. Anat., **81**:52, 1943.

Ito, M.: Studies on melanin: X. Incontinentia pigmenti. Tohoku J. exper. Med., **54**:67, 1951.

Jaramillo, R., Manterola, A., and Rosselot, J.: Incontinentia pigmenti. Rev. chil. Pediat., **19**:654, 1948.

Kawamura: Cited by Kitamura, 1954.

Kitamura, K., Fukushiro, R., and Miyabayashi, T.: Incontinentia pigmenti in Japan. Arch. Derm. & Syph., **69**:667, 1954.

Kühling, F.: Incontinentia pigmenti. Dissertation, Wurzburg, 1949.

Lapiere, S.: Incontinentia pigmenti. Arch. belges. Derm. & Syph., **7**:156, 1951.

Levin, O.: Nevus reticularis and pigmentosus. Arch. Derm. & Syph., **27**:141, 1933.

Loveman, A. B., Fliegelman, M. T., Weidman, A. I., and Rein, C. R.: Incontinentia pigmenti: A report of three additional cases. J. Pediat., **40**:442, 1952.

Miller, O. B., Arbesman, C., and Baer, R.: Disceminated herpes simplex (Kaposi's varicelliform eruption). Arch. Derm. & Syph., **62**:477, 1950.

Moncorps, C.: Incontinentia pigmenti bei einem 2¼ jahrigen Knaben. Ztschr. Haut u. Ges., **50**:381, 1935.

Murrell, T., and Lingamfelter, C.: Personal communication.

Naegeli, O.: Familiärer Chromatophorennavus. Schweiz. med. Wschr., **97**:48, 1947.

Philpott, J. A., Jr., Woodburne, A. R., and Philpott, O. S.: Incontinentia pigmenti. Arch. Derm. & Syph., **71**:214, 1955.

Pierini, L. E., Garcia, L. A., Pomposiello, I., and Rey, O.: Incontinentia pigmenti (Bloch-Sulzberger). Rev. argent. Dermatosif., **29**:181, 1945.

Rein, C. R., and Weidman, A. I.: Incontinentia pigmenti (Bloch-Sulzberger). Arch. Derm. & Syph., **65**:361, 1952.

Scott, J. Graham, Friedmann, A. I., Chitters, M., and Pepler, W. J.: Ocular changes in the Bloch-Sulzberger syndrome (incontinencia pigmenti). Brit. J. Ophth., **39**:276, 1955.

Seidlmayer, H.: Ueber eine neues Syndrom multipler Abartung: Pigmentanomalie, Kongenitale ektodermale Nysplasia, hebenneirenmarkaplasie Ztsch. Kinderh., **63**:451, 1942.

Siemens, H. W.: Die Melanosis Corii Degenerativa, eine neue Pigmentdermatose. Arch. Derm. & Syph. (Berlin), **157**:382, 1929.

Sobel, N.: Incontinentia pigmenti. Arch. Derm. & Syph., **58**:467, 1948.

Sulzberger, M. B.: Ueber eine bisher nicht beschriebene congenitale Pigmentanomalie (incontinentia pigmenti). Arch. Derm. & Syph. (Berlin), **154**:19, 1928.

Sulzberger, M. B., and Bloom, D.: Incontinentia pigmenti. Arch. Derm. & Syph., **58**:468, 1948.

Sulzberger, M. B., Frazer, J. F., and Hunter, L.: Incontinentia pigmenti. Arch. Derm. & Syph., **38**:57, 1938.

Uebel, H., Ludwig, A., and Korting, G.: Zur Kenntnis der Incontinentia Pigmenti (Bloch-Sulzberger). Arch. Derm. & Syph. (Berlin), **190**:114, 1950.

Watanabe: Cited by Kitamura, 1954.

EVALUATION OF THE "PHOSPHENATOR" DEVICE*

FOR THE DETECTION OF INCREASED INTRAOCULAR PRESSURE

LEONARD T. KURLAND, M.D., DAVID SACHS, M.A., AND LARRY C. KERPELMAN
Bethesda, Maryland

AND

F. STERLING DAVIS, JR., CAPT. (MC) U.S.A.
Washington, D.C.

INTRODUCTION

According to recent reports by Humphrey and Murgolo,[1] a low voltage, low amperage electric current applied for a few seconds to the skin bitemporally just behind the eyes "will excite the optic end-organs so that a bluish-white phosphene (light sensation) is observed by the normal eye, while in the case of increased intraocular pressure the patient reports yellow, orange, or red phosphenes." The test is easily and rapidly performed, and "it is not necessary to contact the eye at any time."[1] The advertisement and the brochure, which accompanies the Phosphenator device manufactured by the Hausted Company of Medina, Ohio, state that this is a "new electronic instrument for the rapid diagnosis of glaucoma and macular and peripheral loss of vision."[2]

Since glaucoma is a major ophthalmologic problem in the older population, a method of glaucoma detection which is accurate, simple, and atraumatic would be of obvious advantage. The present investigation was undertaken to determine the validity and reliability of the Phosphenator to select individuals with increased intraocular pressure and to identify persons known to have glaucoma.

METHOD

One hundred and ninety-five male subjects from various wards in the U. S. Soldiers' Home Hospital (group I) and 27 male and 22 female subjects at the Walter Reed Army Hospital (group II) were examined. The median age of Group I was 72 years; that of Group II was 61 years. There were several Negro patients in each group.

Group I consisted largely of elderly patients whose hospitalization was not necessarily related to any ophthalmologic complaint. On the other hand, 36 of the 49 subjects in Group II had glaucoma†; the others were normal subjects or patients with conditions other than glaucoma. The intraocular pressure of the glaucoma patients was well controlled by miotics, surgery, or both in 23 of the 36 patients.‡ Tension fluctuated or was difficult to control in the other glaucoma patients.

The subjects in both Groups I and II were tested by a member of the investigative team using a Phosphenator which was supplied by Dr. Carroll E. Humphrey. The testings were conducted in accordance with the directions in the Phosphenator brochure[2] and after preliminary instruction by Dr. Humphrey. Following a standard pretest introduction, the subject's responses to standard testing questions were recorded for each eye. The following questions were asked:

---

* From the Epidemiology Branch and the Biometrics Branch, National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda, Maryland, and the Ophthalmology Research Unit, Walter Reed Army Hospital, Washington, D.C. An abstract of this paper was presented before the American Academy of Ophthalmology and Otolaryngology, Chicago, Illinois, October 17, 1957.

† The diagnosis of glaucoma on the patients included here had been made after thorough study at the Walter Reed Army Hospital Ophthalmology Department. The diagnosis was reached after routine ophthalmologic examinations as well as the following: (1) Repeated tensions with different weights on different days; (2) visual field examinations; (3) provocative water tests; (4) gonioscopy; (5) where indicated, electrotonography.

‡ The 36 glaucoma patients had been classified as follows: Open angle, 24; narrow angle, seven; secondary, five.

1. What do you see? (If no response, do you see a flickering?)

2. If affirmative, what color is the flickering light in each eye?

3. Select the color from the chart which corresponds closest to the color you saw during the test. The standard color chart prepared by Dr. Humphrey has eight color discs: (1) white, (2) pale blue, (3) pale yellow, (4) bright yellow, (5 and 6) shades of orange, (7 and 8) shades of red. According to Humphrey, color responses 1, 2, and 3 are "normal," colors 4 to 8 are "abnormal responses, indicating increased intraocular pressure."[2,3]

Each subject was then tested for color blindness and color comprehension by a member of the investigative team using standard Ishihara color cards. The subjects in Group I were then examined independently by two members of the investigative team, each using a standardized Schiøtz tonometer. Group II included many patients who had extensive ophthalmologic studies previously and only a single tonometer reading was taken shortly after the Phosphenator and Ishihara tests were carried out for the subjects in that group.

It should be stressed that in every instance the results of the Phosphenator, Ishihara, and tonometer tests were known only to that member of the investigative team who administered the particular test. No information relating to the condition of the eyes was obtained from any of the subjects prior to the recording of the test results.

Results of the various tests were combined for each subject and appropriate statistical evaluations were then carried out.

Results

Most of the subjects, both Groups I and II responded to the Phosphenator tests in the "expected" manner, that is, they gave a straightforward description of the flickering and the color and selected a color from the chart which corresponded to the description. Many other subjects either described a color which was not compatible with that which they selected from the chart, insisted they saw nothing, or were unable to select any color from the chart. Thus, the subjects were categorized, on the basis of these factors, into three categories:

1. *Acceptable* (A). The subjective description of the color sensation produced by the Phosphenator stimulus was in agreement with the color chosen from the color chart.

2. *Questionable* (Q). The subject was undecided and altered his choice of color, with or without repetition of the Phosphenator stimulus.

3. *Unreliable* (U). The subject was unable to describe what he observed or there was gross inconsistency in the subject's response.

The categorization of responses for the subjects in both groups is presented in Table 1. The percentage of acceptable replies was appreciably lower in Group I which included many very elderly and debilitated individuals. It is clear that even a simple test such as the Phosphenator has some limitations for population surveys if it requires comprehension of even elementary directions and a description of a subjective experience.

The "acceptable" (A) subjects were then divided, on the basis of the Ishihara test results, into two classes: color normal (N) and color defective (D). The class whose response to the Ishihara test was unsatisfactory included individuals with poor comprehension of the test as well as color blindness. Thirty-five of the 128 A subjects of Group I and nine of the 43 A subjects of

TABLE 1

CLASSIFICATION OF PATIENTS BY TYPE OF RESPONSE TO PHOSPHENATOR TEST

Category of Response	Group I		Group II	
	Number	Percent	Number	Percent
Acceptable	128	66	43	88
Questionable	21	11	3	6
Unreliable	46	23	3	6
Total	195	100	49	100

Group II were found to be color defective.

Statistical analyses of the results of the Phosphenator test and tonometer reading were then made for the 95 AN ("acceptable" and normal Ishihara test result) subjects in Group I and the 34 AN subjects of Group II. The patient's color selection for each eye following the Phosphenator stimulus was plotted against the corresponding tonometer reading. For Group I, the average of the two tonometer readings was taken since these independent readings were found to agree very well in almost every instance.

The results of the examination of 185 eyes for Group I are summarized in Table 2. In 19 eyes (10.3 percent), the average tension was greater than 25 mm. Hg. In only one of these 19, however, was the Phosphenator response abnormal. In seven eyes the tension was greater than 30 mm. Hg, but in all these the Phosphenator response was the "normal" blue or white. On the other hand, an "abnormal" color response was reported for six eyes but in only one of these was the tension suspiciously high. Re-examination several weeks later of the subjects with either abnormal Phosphenator responses or increased tension failed to affect these results materially.*

On the basis of this analysis, it would appear that the Phosphenator results cannot be depended upon to differentiate subjects with increased tension from those with normal tension.

In the 65 AN eyes of Group II, there were 14 (21:5 percent) with tension above 25 mm. Hg. In none of these was the Phosphenator color response "abnormal" (table 3). Only one patient in the AN group gave an abnormal Phosphenator response. This patient, a 67-year-old man, had chronic open-angle glaucoma. A left iridencleisis four years previously and two-percent pilocarpine four times daily in the right eye had controlled his tension which on the day of this examination was 12 mm. Hg, O.S., and 17 mm. Hg, O.D.

TABLE 2

TONOMETER READING VERSUS COLOR CHART SELECTION FOLLOWING PHOSPHENATOR STIMULUS FOR EACH OF 185 EYES* OF 93 AN PATIENTS IN GROUP I

Average Tonometer reading (in mm. Hg)	Phosphenator Color Response									Total
	Normal			Abnormal						
	1	2	3	4	5	6	7	8		
0– 9	2	—	2							
10–14	4	17	5	1	—	—	—	—		166
15–19	28	38	15	1	—	—	—	—		
20–24	20	22	8	1	—	2	—	—		
25–29	5	5	1	1	—	—	—	—		
30–34	1	3	—	—	—	—	—	—		19
35–39	2	—	—	—	—	—	—	—		
40 and over	—	1	—	—	—	—	—	—		
Total	179			6						185

* One subject was blind in one eye.

TABLE 3

TONOMETER READING VERSUS COLOR CHART SELECTION FOLLOWING PHOSPHENATOR STIMULUS FOR EACH OF 67 EYES* OF 34 AN PATIENTS IN GROUP II

Tonometer Reading (in mm. Hg)	Phosphenator Color Response									Total
	Normal			Abnormal						
	2	2	3	4	5	6	7	8		
0– 9										
10–14	4	3	—	—	1	—	—	—		53
15–19	5	7	3	—	1	—	—	—		
20–24	7	20	2	—	—	—	—	—		
25–29	3	7	—	—	—	—	—	—		
30–34	1	1	1	—	—	—	—	—		14
35–39	—	—	—	—	—	—	—	—		
40 and over	1	—	—	—	—	—	—	—		
Total	65			2						67

* One subject was blind in one eye.

* Of the seven subjects with an abnormal Phosphenator response and normal tension, six were still found to have tensions well within normal limits; their ophthalmologic examinations were negative in all other respects. One of the seven had a tension of 26 mm. Hg in one eye, but his examination was otherwise negative.

Thirteen of the 16 subjects (19 eyes) with suspicious or increased tension were re-examined for glaucoma. Seven were found to have chronic open-angle glaucoma, five were newly diagnosed cases resulting from these tonometry studies; one was considered borderline and suspicious; and five were found to have normal tensions and no other evidence of glaucoma.

Of the remaining 33 AN patients, all of whom gave a normal Phosphenator response, 10 were controls and did not have glaucoma and 23 were patients of the glaucoma clinic of the Walter Reed Army Hospital.

Of these 23 glaucoma patients, 20 had wide-angle glaucoma, one had narrow-angle glaucoma, and two had secondary glaucoma. Tension was well controlled with miotics, surgery, or both in most cases. However, tension was greater than 30 mm. Hg in one patient with a secondary glaucoma and in three patients with wide-angle glaucoma; tension was between 25 and 30 mm. Hg in six other patients with wide-angle glaucoma and in one other patient with secondary glaucoma. In each of these cases with an elevated tension, the Phosphenator responses were normal and equal in the two eyes, even when the tension was elevated in one eye only!

On the basis of this analysis, we find the Phosphenator cannot be relied upon to identify glaucoma patients whether their tensions at the time of the examination are elevated or not.

The results of the comparison of intraocular pressure and the Phosphenator response for the two groups are summarized in Table 4. The over-all frequency of an "abnormal" Phosphenator response was 3.3 percent. There is no difference in the frequency of the abnormal Phosphenator response with respect to intraocular pressure.

TABLE 4

TONOMETER READING VERSUS COLOR CHART SELECTION FOLLOWING PHOSPHENATOR STIMULUS FOR EACH OF 252 EYES OF 127 AN SUBJECTS IN GROUPS I AND II

Tonometer Reading (in mm. Hg)	Phosphenator Color Response		Percent Abnormal
	Normal	Abnormal	
0–19	133	4	2.9
20–24	79	3	3.7
25–29	21	1	4.5 \} 3.0
30 and over	11	0	0.0 \}
Total	244	8	3.3

Further correlations by age, sex, or race fail to add to these disappointing results. It might be stated that for the 44 AD subjects (acceptable, but Ishihara-defective individuals) there was one patient with increased tension who had a normal Phosphenator color response; in this same group there were 13 "abnormal" Phosphenator responses, but in all of these the tension was less than 25 mm. Hg. Seven of this AD group were patients from the Walter Reed Army Hospital glaucoma clinic; all but one of the seven had given a normal response to the Phosphenator test.

Although a tonometer reading above 25 mm. Hg is believed to represent a suspiciously high intraocular pressure, it can be seen from Table 4 that selection of 20 mm., or even 30 mm., Hg, as the point of increased tension gave the same unsatisfactory results for the Phosphenator test.

COMMENT

This study fails to show that the Hausted Phosphenator, as developed by Humphrey, is of value in detecting increased intraocular pressure. This conclusion holds whether the arbitrary selection of normal intraocular pressure is 20, 25, or 30 mm. Hg. The Phosphenator test provided equally poor results in locating subjects with increased intraocular pressure in Group I and identifying patients of the Walter Reed Army Hospital glaucoma clinic.

An appreciable number of subjects failed to give an acceptable color response when tested with the Phosphenator stimulus. Some of these were senile but many elderly subjects who were otherwise well oriented and co-operative could not describe the effect of the stimulus. This emphasizes the need for a screening device for glaucoma which is less dependent upon a subjective response.

In passing, it might be pointed out that, in contrast to the report by Humphrey and Murgolo,[4] we found it difficult to evaluate the field of vision with the Phosphenator device.

There were very few complaints of discomfort when the stimulus was applied, and, to our knowledge, the Phosphenator test is harmless.

Although in our hands the Phosphenator test had no merit as a means of identifying persons with increased intraocular pressure, further study might seem worth while to determine why a few of those tested reported observing yellow, orange, or red phosphenes.

Summary

The Phosphenator developed by Humphrey and now being widely advertised and marketed for the "rapid diagnosis of glaucoma" was evaluated in 195 elderly veterans at the U. S. Soldiers' Home Hospital (group I) and in 49 patients and employees at the Walter Reed Army Hospital (group II). Individuals who gave a poor response to the Ishihara color test and those who could not give a reliable response to the Phosphenator test were not included in the statistical evaluation. The tonometer and Phosphenator results are presented for 93 patients (185 eyes) in Group I and 34 patients and employees (67 eyes) in Group II. The Phosphenator results failed to differentiate individuals with low or suspiciously high intraocular pressures. The Phosphenator response was "abnormal" in seven of the 219 eyes (3.2 percent for the two groups combined) with tension under 25 mm. Hg and in one of 33 eyes (3.0 percent) with a tension above 25 mm. Hg. In 32 of the 33 eyes with high or suspiciously high tension, a normal response to the Phosphenator stimulus was obtained. In Group II, the Phosphenator was not a reliable means of differentiating the normal subjects from glaucoma patients whether the tensions in the patients were well controlled or not at the time of the examination. In individuals with unilocular increased tension, there was no difference in the Phosphenator response for the two eyes.

Conclusions

The Phosphenator was found to be of no value in detecting increased intraocular pressure. Our results have failed to substantiate the claim that the device has merit in the diagnosis of glaucoma since it failed to provide a reliable means of identifying known glaucoma patients. We believe there is a potential danger in the use of this apparatus because it may give a false sense of security regarding the absence of glaucoma to individuals tested by this device alone.

Addendum

The results of a recently published preliminary report by Lipetz[5] are in agreement with those of the present study. Lipetz concludes "that as a test for glaucoma or of intraocular pressure, the Phosphenator test cannot be recommended."

Public Health Service,
National Institutes of Health (14).

Acknowledgments

We wish to express our appreciation for the cooperation of the medical staff of the U. S. Soldiers' Home and Hospital, under the direction of Brig. Gen. H. D. Offutt, U.S.A. (Ret.); Lt. Col. T. L. Hendrix, M.C., U.S.A.; the support of this study by Col. Austin Lowrey, Jr., chief, Ophthalmology Service, Walter Reed Army Hospital; and the technical assistance of Lieut L. Worglen and Lieut. F. Tomlinson, U.S.A., M.S.C.

References

1. Humphrey, C. E., and Murgolo, V. J.: A simple and rapid test for glaucoma. EENT Monthly, **35**: 106 (Feb.) 1957.
2. Hausted Manufacturing Company, Medina, Ohio: Instructions for using the Phosphenator, a new, electronic instrument for the rapid diagnosis of glaucoma and macular and peripheral loss of vision.
3. Humphrey, C. E.: Personal communication.
4. Humphrey, C. E., and Murgolo, V. J.: The use of phosphenes in detecting visual loss. EENT Monthly, **36**:170 (Mar.) 1957.
5. Lipetz, L. E.: Preliminary report: Evaluation of the Phosphenator Test for glaucoma. J. Am. Optometric Soc., **29**:246, 1957.

NOTES, CASES, INSTRUMENTS

A LENS FORCEPS WITH IRIS RETRACTOR

For round-pupil cataract operation

A. Benedict Rizzuti, M.D.
Brooklyn, New York

The round-pupil cataract extraction can be simplified by the proper retraction of the iris when the surgeon employs the "sliding technique" from above.

Description of instrument

To serve this purpose, an instrument is described* that consists of a Verhoeff lens forceps in which there is incorporated a small shelf to act as an iris retractor. The small protruding shelf or ledge is shaped concentrically, hollowed concavely, and placed at the proximal end of one of the ring blades of the lens forceps. The shelf protrudes 1.5 mm., and measures across in its greatest diameter approximately 3.0 mm. The iris ledge is hollowed concavely better to accommodate the iris when it is retracted to simulate the condition that exists in cases where an iridectomy is performed.

Fig. 1 (Rizzuti). Lens forceps with iris retractor.

Method of use

The lens forceps is introduced into the anterior chamber from above with the ring blade containing the iris shelf placed in a prone position. The iris is then gently retracted to its fullest extent while point pressure is applied inferiorly at the corneal limbus with the tip of a muscle hook. When the superior pole of the lens has been titled sufficiently forward, the two rings of the lens forceps are approximated to grasp the lens short of its pre-equatorial area at its superior aspect. The iris can then easily slip over the superior border of the lens. If difficulty is encountered at this time, the heel of the muscle hook can be used to slide the iris over the superior lens pole. The lens is subsequently extracted intracapsularly according to the usual accepted methods.

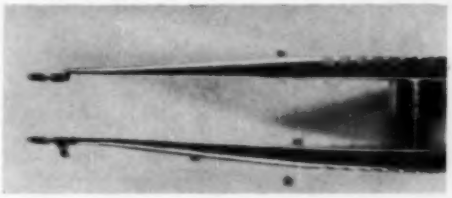


Fig. 2 (Rizzuti). Close-up of iris retractor.

The instrument is simple in construction and not difficult to manipulate. It has a particular advantage to the surgeon when he finds himself having to operate without the proper assistance.

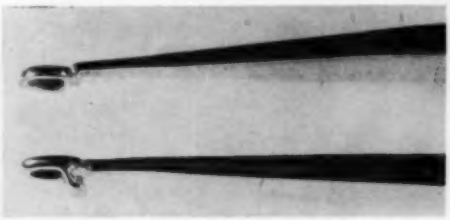


Fig. 3 (Rizzuti). Note concentric and concave lip on lower blade.

An instrument to achieve a similar purpose has been previously designed and reported by me.* Although that instrument proved to be very effective, the best results were obtained when the proper assistance was available.

160 Henry Street (1).

* This instrument may be obtained from the Storz Instrument Company, 4570 Audubon Avenue, Saint Louis 10, Missouri.

* Rizzuti, A. B.: An iris retractor and expressor: For round-pupil intracapsular cataract extraction. Am. J. Ophth., **37**:941 (June) 1954.

CONCOMITANT SQUINT

Method of recording the deviation

J. J. Lijó Pavía, M.D.
Buenos Aires, Argentina

(Translated by David Shoch, M.D., Chicago)

In this paper I present a simple and practical method of recording objectively the deviation in concomitant squint by superimposition photography. It is applicable to all patients with good fixation in the deviated eye.

This photographic record is advantageous in the following types of cases:

1. Concomitant squint with a small angle of deviation.
2. Apparent squint caused by excessive angle alpha in which a cover test is negative.
3. Horizontal squint accompanied by a small vertical component.

This procedure is based on the existence of a constant angle of squint in concomitant strabismus where the primary and secondary deviations are the same. My procedure records photographically, on one film, both positions of the squinting eye. This is done by a variation of the "cover test" (fig. 1).

At the beginning a Leica camera was used but because of difficulties in obtaining good fixation I switched to a Nordenson retinal camera. With this apparatus I can project a bright, uniform bundle of light which holds the full attention of the patient. The subject is placed before the camera with his head in the rest. The camera is focused on the squinting eye and the normal eye fixes a small and shiny cross alongside the camera.

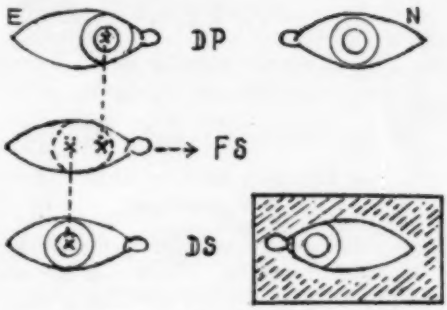


Fig. 1 (Lijó Pavía). Scheme from E. Fuchs (modified).
E = squinting eye; N = normal eye.
DP = primary deviation; DS = secondary deviation (the normal eye is covered with opaque glass).
FS = Scheme of the superimposed photographs with $\dot{x}$ and $\bar{x}$ representing the two white dots in the positives.

An exposure is made (1/10 second) then an assistant covers the normal eye and the squinting eye takes up fixation on the beam from the camera. A second exposure is then made on the same film (also at 1/10 second). The slightest deviation in the optical axes will result in a positive which shows two white points which are separated by an amount corresponding to the deviation of the eyes. My original film has a 30-mm. diameter and the positive is enlarged to 50 mm. From this an equivalence can be established of millimeter separation to degrees of deviation.

Case reports

Case 1

M. E. G., an eight-year-old girl, had an inward deviation of the left eye from the age of three years. She had had complete occlusion of the right

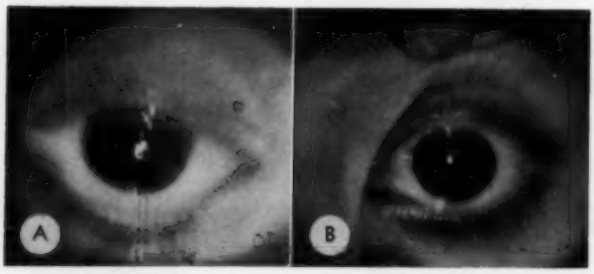


Fig. 2 (Lijó Pavía). (A) Superimposed photograph of Case 1 before operation. White dots separated 1.25 mm. (B) Superimposed photograph Case 1 after operation. Single white dot indicates parallelism.

Fig. 3 (Lijó Pavía). Photograph of Case 2 of both eyes preoperatively shows left esotropia.

eye for one year. Examination showed a bilateral hyperopia of 5.0D., and left esotropia of 15 degrees with normal fixation. Vision in the right eye was 10/10; left eye, 5/10. Occlusion of the right eye was ordered. Six months later the findings were the same and the superimposed photograph (fig. 2-A) was made and operation advised. The left medial rectus was recessed five mm. After 48 hours, the dressing was removed from the left eye. The right eye was kept closed for 10 days except for stereoscopic exercises for one-half hour twice daily. Two months later the superimposed photograph (fig. 2-B) was obtained showing a parallelism of the eyes. Vision in the left eye was 7/10 and stereoscopic exercises were continued.

CASE 2

D. M. K., an eight-year-old girl, had a left esotropia present from age of three years. Examination showed a bilateral hyperopia of 2.0D., and a left esotropia of 30 degrees (fig. 3). There was normal fixation with the left eye and vision was: right eye, 8/10, and left eye, 4/10. The superimposed photograph (fig. 4-A) was taken and surgery advised. A recession of the left medial rectus and a three-mm., advancement of the left lateral rectus were done.

Following surgery the right eye was occluded and two weeks later the vision in the left eye was 5/10. Steroscopic exercises were ordered and one month later a superimposed photograph was made (fig. 4-B) which showed parallelism of the eyes.

SUMMARY

A simple procedure has been described which is useful not only for recording the deviation but for postoperative evaluation as to the benefits to be derived from orthoptic training.

Avenida Quintana, 104.

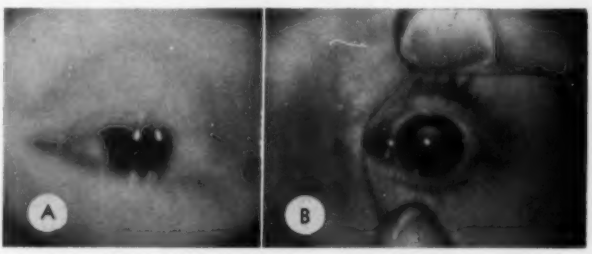


Fig. 4 (Lijó Pavía). (A) Superimposed photograph of Case 2 before surgery. White dots separated 4.0 mm. (B) Case 2 after surgery. Only one white dot is seen, indicating parallelism.

FLUORESCENT LAMP EXAMINING UNIT

COMBINING WHITE AND BLACK LIGHT

LESTER STEIN, M.D.
Steubenville, Ohio

The average illuminating unit such as the Burton Hand Magnifier[1] is now constructed with electrical circuits to use only one pair of four-watt fluorescent lamps[2] at a time. It is cumbersome to change the lamps from white light to the black light[3] type so as to provide rapid alternation of light for intensive study of luminescence of the ocular tissues.

In my work on determination of precancerous alteration of the ophthalmotissues as demonstrated by changes in luminescence of the anterior ocular segment and the ocular adnexa, I found that a simple reconstruction of the Burton Unit will provide a most efficient method of incorporating and utilizing an extra set of miniature bi-pin sockets, ballast, and switch so that a double pair of fluorescent lamps can be incorporated in the unit and alternation of white light and Woods light is facilitated.

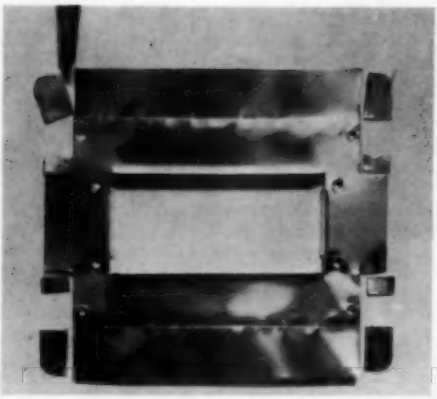


Fig. 1 (Stein). Revision of aluminum reflector from Burton Magnifier unit.

Directions for construction

The Burton Unit is completely disassembled by unscrewing the four screws at each end of the magnifying lens which holds the aluminum reflector to the Bakelite casing. The bi-pin sockets are also unscrewed and unwired. With a metal shear the aluminum reflector is cut at each end to permit mounting of an additional pair of miniature bi-pin sockets as shown in Figure 1. Holes are drilled for these sockets to be fastened in position. This must be done carefully while positioning a black light tube to determine adequate clearance. Thus two pairs of tubes, a white light outer pair on the original circuit and a black light pair on a new similar circuit are incorporated (fig. 2).

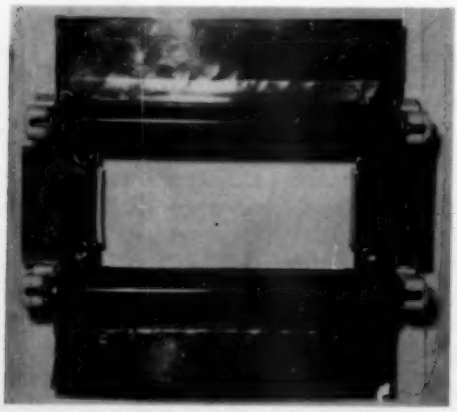


Fig. 2 (Stein). Black light lamps in position.

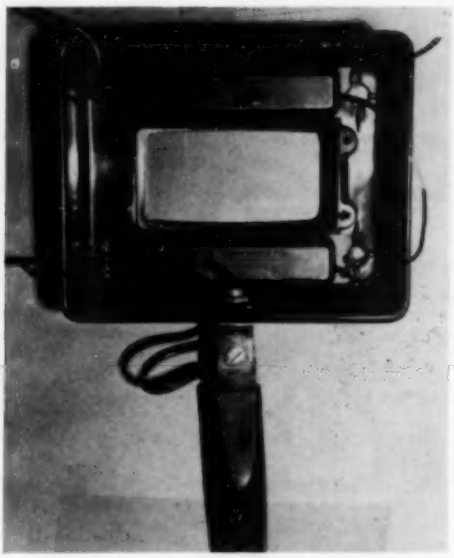


Fig. 3 (Stein). Robertson ballasts in Bakelite housing.

It is necessary to purchase another eight-watt ballast[4] and an additional single or double-button Gaynor switch for the series hook-up of the paired lamps. The Bakelite casing of the Burton Unit is drilled appropriately as shown in Figure 3; thus, two holes at each end of the casing are obtained and

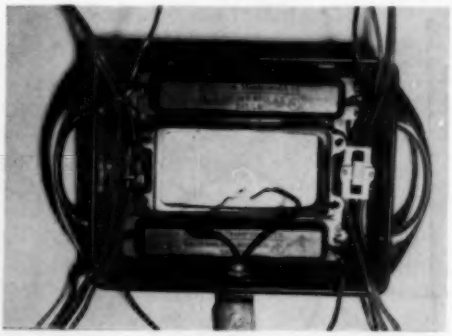


Fig. 4 (Stein). Position of Gaynor switches at each end.

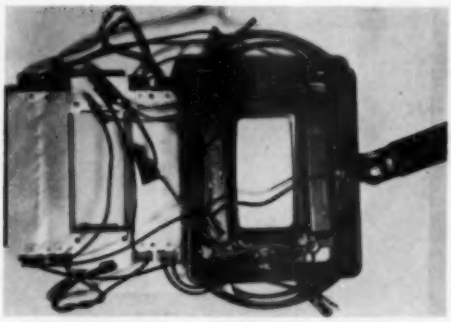


Fig. 5 (Stein). Loose wiring of sockets, ballasts switches, and main line prior to test run. After testing, the leads are shortened, soldered and taped. The lens is replaced and the reflector fastened into place.

Fig. 6 (Stein). Burton unit on Fleximatic support for use in chair diagnosis.

two Gaynor[5] switches mounted; one for each pair of fluoro-lamps as depicted in Figure 4. The complex wiring is fitted in the narrow confines as in Figure 5 and, after testing, the reflector is fastened back over the lens to finish the job.

This set-up permits rapid alternation of white light and black light flux without disturbing the mesopic state of the observer. After a few minutes of dark adaptation, I light up the black light pair of lamps first and then with my other hand on the white light button illuminate the white light pair to catch quick glimpses, alternating with black light luminescence. A binocular magnifier loupe is worn for additional magnification while peering through the magnifying lamp. Mounting the unit on the Burton Fleximatic support lends mobility for chair diagnosis as depicted in Figure 6.

203 Sinclair Building.

References

1. Burton Manufacturing Co., Colorado Ave. at 26th St., Santa Monica, Calif.

2. Four watt, T5, white fluorescent lamp made by General Electric Co. or Westinghouse Co. The nominal length of this lamp is 6 in. The width is 5/8 in. It has a bi-pin miniature base. The electrical characteristics are 35 volts, 0.125 amp., the illumination, 2,100 lamberts.

3. Black light lamp or BLB type, four-watt integral filter made by General Electric Company.

4. Ballasts obtainable from Stratfield Co., 434 Grand St., Bridgeport 4, Conn., model S1-108.
Robertson Transformer Co., Blue Island, Ill., L-8 model, eight watts.
Bi-pin miniature sockets from Kulka Electric Co., Mount Vernon, N.Y., catalog #570.

5. Gaynor switches obtainable from Edwin Gaynor, 68 Church Hill Road, Bridgeport 4, Conn. The one-button push switch is catalog #6551, the two-button switch is catalog #8242-25.

IMPROVED PTOSIS OPERATION WITH BURIED SILK SUTURES*

KOHEI OHASHI, M.D.
Tokyo, Japan

During recent years, there have been interesting attempts to correct ptosis with subdermal sutures. Most of these procedures have failed for one or another of the following reasons:

Either the procedure was not so easy to perform, there was postoperative loosening of the sutures, due to difficulty in passing them subdermally; or there was a tendency for the lid margin to become pointed after application of a single loop.

The method herein described is a modification of the Friedenwald-Guyton technique. I have now employed it in 20 cases with no failures. It is easy to perform. Important to remember is that fixation of the suture is attained by use of the frontal periosteum and tarsal tissue. Komoto's or Guyton's suture provides permanent correction. My technique is:

After inserting a spatula, a supraciliary incision (fig. 1-A, B, and C) and a lid-margin incision (fig. 1-D and E) are made. One end of a loop suture threaded through a large curved surgical needle is passed subdermally from C to B (fig. 1), scooping the frontal periosteum. Thereafter, this needle is advanced from B to E (fig. 1). The other

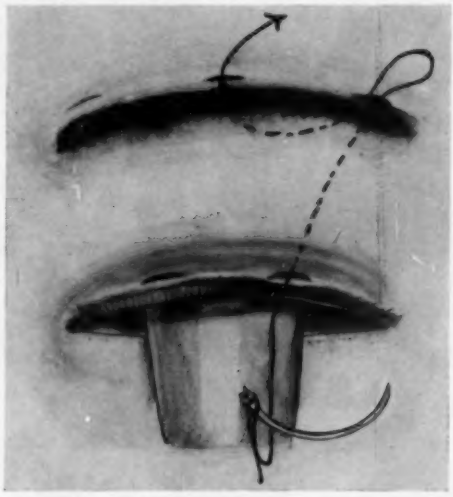


Fig. 2 (Ohashi). One loop suture is passed beneath the palpebral skin with surgical needles.

Fig. 1 (Ohashi). The skin incision is made.

Fig. 3 (Ohashi). Two loop sutures are just passed out from wounds D and E of lid margin (fig. 1).

* From the Department of Ophthalmology, Tokyo Jikei-kai School of Medicine, Minato-ku.

Fig. 4 (Ohashi). One end of loop is scooped through the tarsal tissue with a small needle.

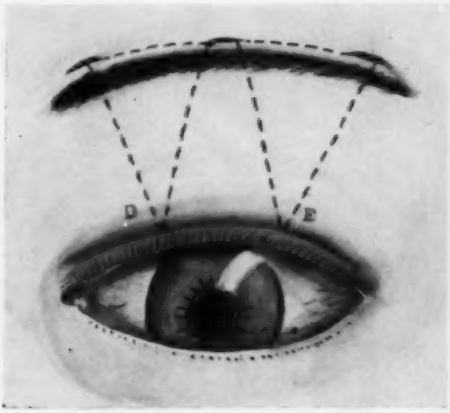


Fig. 5 (Ohashi). Stage in the operation when the sutures have been knotted and cut.

end of the loop is also passed to E. (See Figure 2.) Another loop suture is similarly employed, as in A, B, and D (fig. 1). Thereafter the sutures are unthreaded (fig. 3). After grasping the flap edge of E or D (fig. 1) with forceps, one suture end, threaded through a small needle, is passed horizontally through the deep tissue. Controlled by a dilated lid fissure, these two ends are knotted three times. Avoid unfastening this knot, but trim the ends (fig. 4).

Since the suture ends are buried subdermally under the skin, wound-closure sutures are not necessary (fig. 5). After one to two weeks, the ptosis is satisfactorily corrected in every case.

Department of Ophthalmology,
Tokyo Jikei-kai School of Medicine.

REFERENCES

1. Friedenwald, J. S., and Guyton, J. S.: Am. J. Ophth., **31**:411, 1948.
2. Johnson, C. C.: Am. J. Ophth., **38**:129, 1954.
3. Ohashi, K.: Rinshoganka (Japanese) **9**:1517, 1955.

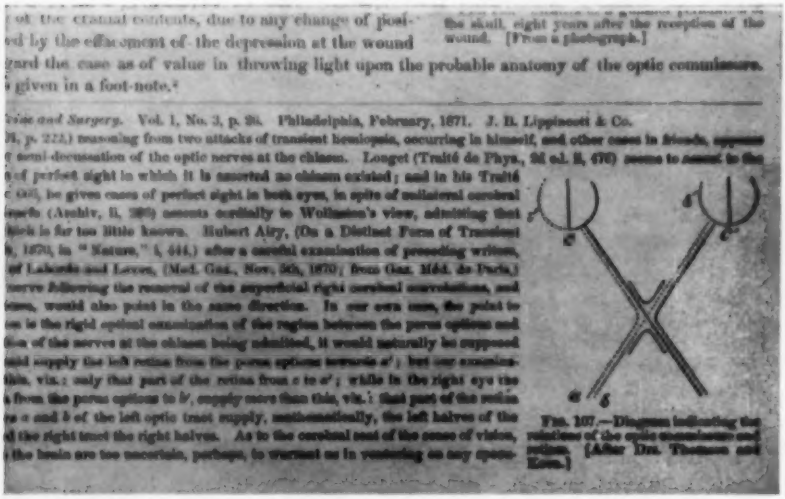
INTERESTING MEDICAL RECORDS ON THE OPTIC CHIASM

W. J. MARSHALL, M.D.
Springfield, Missouri

A number of volumes, published under the direction of the Surgeon General, Joseph K. Barnes, United States Army, were prepared shortly after the Civil War. The title of these were *The Medical and Surgical History of the War of the Rebellion (1861-65).* The second issue was published in 1875. I don't know how many books there were among these publications.

The item referred to here is listed as "Part 1: First Surgical Volume."

Beginning on page 206 is the case history of Private Patrick Hughes, Co. K, 4th New York Volunteers, aged 23 years. Private Hughes was injured at the battle of Antietam on September 17, 1862. His injury is thought to have been the result of a conoidal bullet, entering the right parietal region and



of the cranial contents, due to any change of position ... the skull, eight years after the reception of the wound. [From a photograph.]

ed by the enlargement of the depression at the wound

gard the case as of value in throwing light upon the probable anatomy of the optic commissure. ... given in a foot-note.*

...rgie and Surgery. Vol. I, No. 3, p. 26. Philadelphia, February, 1871. J. B. Lippincott & Co.

...H, p. 222,) reasoning from two attacks of transient hemiopsia, occurring in himself, and other cases in friends, appears ... semi-decussation of the optic nerves at the chiasm. Longet (Traité de Phys., 2d ed. ii, 476) seems to assent to the ... of perfect sight in which it is asserted no chiasm existed; and in his Traité ... ait, he gives cases of perfect sight in both eyes, in spite of unilateral cerebral ... (Archiv, ii, 286) assents cordially to Wollaston's view, admitting that ... which is far too little known. Hubert Airy, (On a Distinct Form of Transient ... 1870, in "Nature," i, 444,) after a careful examination of preceding writers, ... of Laborde and Leven, (Med. Gaz., Nov. 5th, 1870; from Gaz. Méd. de Paris,) ... nerve following the removal of the superficial right cerebral convolutions, and ... cases, would also point in the same direction. In our own case, the point to ... on is the rigid optical examination of the region between the porus opticus and ... tion of the nerves at the chiasm being admitted, it would naturally be supposed ... would supply the left retina from the porus opticus between a' ; but our examina... this, viz.: only that part of the retina from c to a' ; while in the right eye the ... from the porus opticus to b', supply more than this, viz.: that part of the retina ... a and b of the left optic tract supply, mathematically, the left halves of the ... the right tract the right halves. As to the cerebral seat of the sense of vision, ... the brain are too uncertain, perhaps, to warrant us in venturing on any specu...

FIG. 107.—Diagram indicating the relations of the optic commissure and optics. [After Drs. Thomson and Leven.]

Fig. 1 (Marshall). Photograph of diagram appearing in *The Medical and Surgical History of the War of the Rebellion (1861-65).*

emerging from the left occipital. The case history report is rather interesting in numerous respects. It indicates that the man was conscious enough to remove himself from the field of battle and report to a hospital station. He had, seemingly, never lost consciousness or a rational state to any very serious degree or for any period of time.

My particular interest in the case from an ophthalmologic standpoint starts with a notation as to examination by Dr. William Thomson and Dr. W. W. Keen of Philadelphia, who saw Private Hughes on December 20, 1870. This examination was, seemingly, relative to pension applications, and the physicians were obviously much interested in the loss of vision in the patient's right eye. They, seemingly, made a rather complete report of their findings, and it is listed as appearing at that time in *Photographic Review of Medicine and Surgery* (Philadelphia, Lippincott, 1871, v. 1, no. 3, p. 26).

Figure 1 is a photographic copy of the diagram as it appeared in the *Medical and Surgical History* volumes. The notes* were footnotes indicating the interest in the optic pathway. I interpret the interest as pointing out the fact that the optic fields, in the physicians' opinion, are split in the region of the macula, rather than at the plane of the disc. It is also rather interesting to note, in rummaging through these volumes, that there was as much, or more, reliance on artists' illustrations than photographic illustrations. Many of the photographs are supplemented with water color additions.

305 Professional Building.

* "Wollaston (Phil. Trans. 1824, p. 222) reasoning from two attacks of transient hemiopsia, occurring in himself, and other cases in friends, appears to have been the first to point out the semi-decussation of the optic nerves at the chiasm. Longet (Traite de Phys., 2d ed., ii, 476) seems to assent to the explanation, though he refers to cases of perfect sight in which it is asserted no chiasm existed; and in his Traite d'Anat. et de Phys. du Syst. Nerv., p. 666, he gives cases of perfect sight in both eyes, in spite of unilateral cerebral atrophy or traumatic lesion. Von Graefe (Archiv. ii, 286) assents cordially to Wollaston's view, admitting that he proposes nothing new, but that which is far too little known. Hubert Airy (On a distinct form of transient hemiopsia, Proc. Roy. Soc., Feb. 17th, 1870, in "Nature," i, 444), after a careful examination of preceding writers, also supports it; and the experiments of Laborde and Leven (Med. Gaz., Nov. 5th,

BUFFALO-BERRY THORN IN THE EYE

H. D. Harlowe, M.D.
Miles City, Montana

Foreign bodies in the eye are not infrequent or uncommon, but occasionally unusual ones occur. This case and its fortunate outcome appear worthy of reporting.

Report of case

History. The patient, a white boy, aged eight years, was brought to the hospital by his parents. They stated that two and a half hours prior to admission the boy was playing and accidentally ran a thorn into his left eye. They were intelligent enough not to try to remove the thorn and the child had made no attempt to remove it either. It was necessary for the patient to travel a distance of 125 miles in this condition.

Examination revealed a long buffalo-berry thorn (1.5 inches) penetrating the left eye, at approximately the 5-o'clock position. All other physical findings were within normal limits.

Treatment and course. The child was taken immediately to the operating room where a general ether anesthesia was administered. A 6-0 silk pursestring suture was placed around the puncture wound in the eye. The thorn (fig. 1) was then very gently withdrawn from the eye and, at the same time, the pursestring suture was closed. Only a very slight amount of vitreous was lost during the procedure. The wound was carefully inspected and cleansed. Several interrupted silk sutures were then placed to reinforce the injury site. Ophthalmic ointment and an eyepatch were applied to the injured eye. Antibiotic therapy was carried out for 10 days postoperatively.

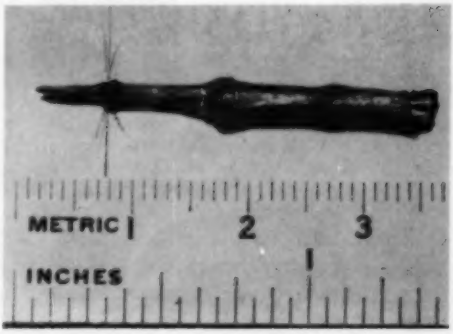


Fig. 1 (Harlowe). Photography of buffalo-berry thorn removed from eye.

Two months later the wound was healed. Examination of the eye revealed the lens to be clear, the fundus normal, and there was no evidence of any retinal detachment. Vision in this eye was found to be normal.

Comment

This case illustrates what severe injury an eye may sustain and yet, with proper management and antibiotic therapy, retain normal vision.

Garberson Clinic.

1870; from Gaz. Med. de Paris), who found atrophy of the right optic nerve following the removal of the superficial right cerebral convolutions, and without any apparent irritative processes, would also point in the same direction. In our own case, the point to which we desire to call special attention is the rigid optical examination of the region between the porus opticus and the macula lutea. The semidecussation of the nerves at the chiasm being admitted, it would naturally be supposed that the fibers *a* from the left tract would supply the left retina from the porus opticus toward *a'*; but our examination shows that it supplies less than this, viz.: only that part of the retina from *c* to *a'*; while in the right eye the fibers *b*, instead of supplying the retina from the porus opticus to *b'*, supply more than this, viz.: that part of the retina from *c'* to *b'*. In other words the fibers *a* and *b* of the left optic tract supply, mathematically, the left halves of the two retinas from *c* to *a'* and *c'* to *b'*, and the right tract the right halves. As to the cerebral seat of the sense of vision, the amount and depth of the injury to the brain are too uncertain, perhaps, to warrant us in venturing on any speculations as to its locality."

OPHTHALMIC RESEARCH

EDITED BY FRANK W. NEWELL, M.D.

Abstracts of papers presented at the meeting of the Eastern Section of the Association for Research in Ophthalmology, at the National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda, Maryland, January 17 and 18, 1958.

Human rhodopsin. George Wald and Paul K. Brown, The Biological Laboratories of Harvard University, Cambridge 38, Massachusetts.

Human rhodopsin in aqueous solution has λ_{max} 493 mμ, lower in the spectrum therefore than the rhodopsins of all other known vertebrates with the exception of certain deep sea fishes. Its molar extinction is 40,000 ± 800. Like other rhodopsins, it bleaches to a mixture of opsin and all-*trans* retinene, and is re-synthesized by incubating opsin with neo-*b* (11-*cis*) retinene. The regenerated rhodopsin has the same λ_{max} as originally; this is due, therefore, not to an unusual retinene, but to a characteristic human opsin. The regeneration in solution from opsin and neo-b retinene is a second-order reaction with a half-time at 29.5°C. of about 2.5 minutes. This is much faster than the synthesis of rhodopsin in living human eye, and than human rod dark adaptation; the rate of both processes in vivo must be limited by reactions which precede the union of neo-b retinene with opsin, the final step in rhodopsin synthesis. In the rods, rhodopsin is virtually in the solid state—highly oriented in close relation with other highly oriented molecules. In this situation its spectrum is displaced toward the red (λ_{max} 500 mμ) and is narrower than in solution. For light entering the rods axially, rhodopsin has also a considerably increased extinction, some 1.5 times higher than when randomly oriented. The spectrum of rhodopsin in rods agrees well in form and position with the spectral sensitivity of human rod vision, measured at the retinal surface.

The mechanism of bleaching of rhodopsin. Ruth Hubbard, Ph.D., and Allen Kropf, Ph.D., The Biological Laboratories of Harvard University, Cambridge 38, Massachusetts.

Rhodopsin, a red visual pigment of vertebrate rods, is composed of a colorless protein, opsin, combined with a hindered *cis* isomer of retinene, named neo-*b* (11-*cis*). Upon exposure to light, rhodopsin bleaches over the orange-red intermediates, lumi- and meta-rhodopsin, to all-*trans* retinene and opsin. The time relations of these processes make it clear that visual excitation must depend upon the conversion of rhodopsin to lumi- or at most meta-rhodopsin.

We have recently found that the primary effect of light on rhodopsin is to isomerize its neo-*b* color group to the all-*trans* configuration. The all-*trans* retinene, still attached as chromophore to opsin, constitutes lumi- and meta-rhodopsin. However, opsin is unable to form a stable chromoprotein with all-*trans* retinene, so that meta-rhodopsin decomposes at temperatures above −15°C. into all-*trans* retinene and opsin.

Light, having converted rhodopsin to lumi- or meta-rhodopsin, at temperatures below −15°C., can go on to isomerize their all-*trans* chromophores to steady-state mixtures of stereoisomers of retinene, all still attached to opsin. The neo-*b* fraction of this mixture constitutes rhodopsin; the iso-*a* fraction, isorhodopsin. On warming, the other isomers leave the opsin surface, and hence constitute

the fraction which bleaches to retinene and opsin.

For the process of vision, the eye has therefore selected a reaction in which light affects directly the *shape* of a protein chromophore. By coupling this with the spatial bequirements of a protein surface, it has provided a mechanism for translating the absorption of quanta into a physiologic event.

The visual system of the honeybee. Timothy H. Goldsmith, B.A., The Biological Laboratories, Harvard University, Cambridge 38, Massachusetts.

The insects are the major group of animals possessing prominent eyes whose visual chemistry is still completely obscure

Retinene$_1$ has now been extracted from the heads of honeybees (apis mellifera). The retinene is apparently bound to protein, from which it can be extracted only with the aid of such polar organic solvents as acetone. About 0.2 μg. have been extracted per gm. fresh weight of heads. This corresponds to slightly more than $3 + 10^{-6}$ micromols per eye, which, per area of retina, is about five percent the amount of visual pigment found in cattle.

A photosensitive pigment also has been extracted from bees' heads. Unlike all visual pigments previously examined, it does not require a solubilizing agent such as digitonin for extraction. A buffer extract of bees' heads, however, contains many colored substances stable to light, which prevent the direct spectrophotometric measurement of the small amount of photosensitive pigment present.

The colored impurities have been largely removed by differential precipitations. The capacity to liberate retinene to polar organic solvents was used as a guide in designing this procedure. The photosensitive retinene complex remains in the solution in 45-percent saturated ammonium sulfate, which precipitates many colored impurities. The photosensitive complex itself is precipitated by 60-percent saturated ammonium sulfate, leaving other colored impurities in solution. It is also soluble in 25 volumes percent ethanol at −12°C., which precipitates still other contaminants.

Through such procedures about 40 percent of the retinene originally present in the heads can be concentrated in a partially purified preparation. This final solution contains a photosensitive pigment with a λ_{max} about 440 mμ. An extract from 220 heads concentrated in 1.0 ml. displays a change of extinction on bleaching of about 0.2. Since about half the retinene complex originally present is lost during the procedure, the amount of photosensitive pigment in the bees' eyes is probably at least twice as great.

As the pigment bleaches in the light, a product is formed with λ_{max} about 370 mμ, which appears to be retinene. The spectrum is somewhat distorted at short wavelengths. This product, estimated as retinene, is produced in about the correct equivalent amount if the molar extinction of the photopigment is taken to approximate that of rhodopsin, about 40,000.

The spectral sensitivity of the compound eyes and ocelli of bees is being measured with the electroretinogram. Evidence has been obtained of a peak of photosensitivity in the compound eye of the drone at about 440 mμ. This peak may be based upon the absorption characteristics of the pigment which has been extracted. The visual sensitivity measurements suggest, however, that this is not the only photosensitive pigment which the bee possesses. It should be recalled that there is good behavioral basis for the assumption that the bee possesses color vision; and it is possible that the 440 mμ pigment is only one of a group of photosensitive pigments concerned with vision in the bee.

Photopic retinal receptors of the typical achromat. Louise L. Sloan, Ph.D., Laboratory of Physiological Optics, Wilmer Institute, The Johns Hopkins Hospital, Baltimore 5, Maryland.

The dark adaptation curve of the typical achromat has two sections, suggesting that his retina has not only normal rods but also a second receptor mechanism which functions in the light adapted eye. It has been variously suggested that these receptors are (a) high threshold rods, (b) normal cones too few in number to provide good acuity and color discrimination, or (c) "blue" cones.

The dark adaptation curves of five achromats were determined employing alternately white (Illuminant C) and blue (Illuminant C plus Wratten 47) test lights to measure the threshold. The increase in threshold when the blue filter is interposed should be 1.6 log units for normal cone receptors, 0.8 for rods, and about 0.4 for blue-sensitive receptors with maximum sensitivity of 440 mμ.

In the second or rod section of the adaptation curves two normals and five achromats showed the predicted increase in threshold of 0.8 log units. In the first section the increase was 1.3 and 1.5, respectively, for the two normals, 0.8 for each of the five achromats. The results suggest that both receptor mechanisms of the achromat have the spectral sensitivity of normal rods.

Eye movements and visual pathways in the chameleon. Magdolna Irányi, M.D., Donald L. Miller, A.B., and Robert B. Livingston, M.D., Basic Research Program, NIMH-NINDB, National Institutes of Health, Bethesda, Maryland.

The chameleon represents an extreme development among land vertebrates of ocular motor independence, and it depends importantly on vision in hunting prey. This interesting reptile has completely independent control of its eye movements; that is, it can look with each turretlike eye at separate targets at the same time. It also has a remarkable excursion of eye movements, approximately 180 degrees vertically and horizontally. In opticokinetic nystagmus, chameleon eye movements are independent in rhythm. In vestibular nystagmus, the eye movements are rhythmically linked together. When a chameleon is getting set to capture some insect, both eyes are directed ahead upon the target, and a swift flick of its long tongue effects capture. The distance may be as far from the jaws as the length of the chameleon's own body. The chameleon strikes an insert target with its tongue with apparently equal facility and accuracy even after one eye has been removed.

There are four recti and two oblique extraocular muscles attached to each globe. The IIIrd, IVth and VIth cranial nerve nuclei are disposed in the same general anatomic pattern in the brainstem as are the analogous structures in man. The medial longitudinal fasciculus is prominent and interconnects the eye motor nuclei. Also, as in man, each of the oculomotor nuclei sends motor fibers to muscles in both orbits. In an animal with eyes moving in opposite directions, the "programming" of motoneuron discharge within each oculomotor nucleus and among the six nuclei together must be very complex. The complete independence of eye movements and the apparently typical muscular and motor nuclear organization imply that the eye motor nuclei in the chameleon cannot be interconnected in some simple reciprocal scheme.

The optic chiasma is entirely crossed. The ventral optic tract fibers go directly to an optic nucleus in the tegmentum, immediately anterior to the exit of the oculomotor nerve. This nucleus is in turn associated with the eye motor nuclei, both directly and indirectly. The main part of

the optic tract sends collateral fibers to the thalamus and ends by forming a roof over the optic lobe. Input into the optic lobe involves thalamic projections as well as the optic tract fibers just described. Neurons residing in the optic lobe distribute dendrites in a radial fashion up into the terminating axons of thalamic and retinal origin. They send their axons down into the brainstem where they undoubtedly influence the eye motor nuclei and motor centers relating to the head, tongue, limbs, and tail. Thus there is a fairly direct visual pathway to an optic nucleus in the tegmentum of the brainstem and a more circuitous route by way of the thalamus and the optic lobe.

The optic lobe is thought to be comparable to the ventral part of the lateral geniculate body in man. There are no "higher" visual centers in the chameleon. Nature provides an example in the chameleon of a relatively simplified neuronal system that is nevertheless capable of integrating visual and eye motor impulses as necessary for tracking moving targets and for establishing the spacial co-ordinates necessary to capture. We are attempting to establish, by a combination of behavioral, neuroanatomic and neurophysiologic techniques, which elements of this system are essential for part or all of these performances.

Electrically induced movements of the iris versus physiologic reflex responses. Otto Lowenstein, M.D., and Irene E. Lowenfeld, Ph.D., College of Physicians and Surgeons, Columbia University, 635 West 165 Street, New York 32.

Iris movements elicited in conscious animals by physiologic stimuli are integrated reflexes. As in other reflex movements, active innervation of the agonist is combined with inhibition of the motor nucleus of the antagonist. In contrast, electrical stimulation of sympathetic or of parasympathetic motor nerves to the iris evokes incomplete motions which differ in speed and dynamic structure from the physiologic reflex pattern. The peak speed of the iris movements evoked in anesthetized animals by electric stimulation of the cervical sympathetic, or of the ciliary ganglion, falls far short of the velocity achieved during pupillary reflexes to light or to sensory stimulation in conscious animals.

For example, when the cervical sympathetic is electrically stimulated with maximal intensity, the resulting iris motion is maximal in extent but relatively slow compared to physiologically elicited reflex dilatation of the pupil. It appeared likely that the parasympathetic-inhibitory factor present in physiologic pupillary reflex dilatation but absent in responses to electrical stimulation was responsible for this difference in peak velocity. To prove this hypothesis sensory stimuli, known to inhibit the third nerve nucleus in narcotized animals, were given simultaneously with the sympathetic stimuli. The iris motions resulting from such combined sensory and sympathetic stimulation were found to be similar in all features to physiologic reflex dilatation of the pupil.

Therefore, electrical stimulation of the agonist and simultaneous electrically produced inhibition of the antagonist combine to give rise to an integrated motion similar to the natural reflex movement. The different types of iris responses were demonstrated and the physiologic significance of inhibition and facilitation, as shown in these reactions, were discussed.

Role of centrifugal inhibitory fibers on the amplitude of the electroretinogram. Jerry H. Jacobson, M.D., and Gidon F. Gestring, Department of Electrophysiology, New York Eye and Ear Infirmary, Albert Einstein College of Medicine, New York.

Various attempts have been made in the past to analyze the complex electro-

retinogram of the mammal. In this report an inhibitory component, not hitherto described in the literature, is demonstrated.

In a series of experiments on cats and monkeys under Pentabarbital anesthesia a unilateral section of the optic nerve was performed about one mm. below the optic chiasm while monitoring the electroretinogram bilaterally. An immediate increase in the amplitude of the electroretinogram in the denervated (isolated) eye was observed which persisted throughout the experiment. Intravenous administration of Hexamethonium and central nervous system stimulants, such as Strychnine and Metrazol, reduced the electroretinogram b-wave amplitude, while central nervous system depressants, such as Nembutal increased the response in the intact eye. Simultaneously, no effect was noticed on the isolated eye. Electrical stimulation of the distal end of the sectioned nerve caused a decrease in the isolated electroretinogram without affecting the intact eye.

It is concluded that inhibitory fibers connect a cephalic inhibitory center with the retina through the optic nerve which functions to depress the amplitude of the electroretinogram. The electroretinogram hence is not a pure retinal response but a summation of the pure retinal response and the effect on it is of a central process. This potential can be abolished with deep anesthesia or optic-nerve section.

The interrelations between critical flicker frequency, the electroretinogram, photochemical mechanisms, and perceptual responses. R. H. Peckham, Ph.D., and W. M. Hart, M.D., 8218 Wisconsin Avenue. Bethesda, Maryland, Sibley Memorial Hospital of American University, Washington, D.C.

Critical flicker frequency is the rate of alternation of lightness and darkness of the stimulus at which perception of alternation disappears and the field seems uniform. When critical flicker frequency is discrete, measurement of the essentially random nature of the phenomenon is ignored, and the estimate, being unstable, loses its correlation to other retinal phenomena. Light on the retina initiates a reversible photochemical reaction which includes specific electrical discharges in individual receptor cells. Integrated, these are found in the electroretinogram. At threshold intensities the perception aroused is a probability function. When the field is flickering the retina is operating in this probability range, and the perception uniquely reflects this randomness. Estimates of critical flicker frequency must be obtained within this probability matrix. When the rate of alternation is constant, estimates of critical flicker frequency can be expressed as that contrast at which 50 percent of the presentations will arouse "flicker" and 50 percent "fusion." Although the perception is random, the measurement is an exact and stable quantity. Correlation of critical flicker frequency with age, and the statistical evaluation was presented. Failure of correlation with the other primary evidences of retinal response may now be avoided. Age, hypertension, glaucoma and dark adaptation, can be investigated. Studies of field defects, the effect of drugs on vision, or ophthalmic protection in operating high speed aircraft can be undertaken, using this sharpened tool for ophthalmic diagnosis.

A technique of measuring the electrical potential across the living rabbit cornea. Anthony Donn, M.D., Department of Ophthalmology Research, College of Physicians & Surgeons, Columbia University, 630 West 168th Street, New York 32, New York, and the Institute of Ophthalmology, 635 West 165th Street, New York 32.

A technique is described by which an

isolated rabbit cornea is mounted between two lucite chambers. When both sides of the cornea are perfused with an artificial aqueous solution, the cornea is able to maintain its normal hydration without swelling. If the preparation is cooled the cornea swells, but when the temperature is raised to 37°C., the cornea gets thinner.

A potential difference of up to 40 millivolts is measured across this mounted cornea. The potential varies in size during the course of six hours; however, the direction of the potential is always constant. The concave side of the cornea is always positive with respect to the convex surface.

The distensibility of the human eye. F. J. Macri, Ph.D., T. Wanko, M.D., and P. A. Grimes, B.A., Ophthalmology Branch, NINDB, National Institutes of Health, Bethesda, Maryland.

Nine human eyes were used in this study. Known volumes of fluid were injected into the anterior chamber and the induced pressure changes in this chamber were recorded manometrically. The rigidity of the eyes was expressed by the equation of Friedenwald,

$$K = \frac{\log P_2 - \log P_1}{\Delta V},$$

and by a modification of the bulk Modulus, $E = \frac{P_2 - P_1}{\Delta V}$, K and E — rigidity, P_2 — final pressure, P_1 — initial pressure, ΔV — volume change. Rigidities were not found to be constant at various intraocular pressure levels with either method of determination. The rigidities were also found to vary as a function of time elapsed between death and the experimental determinations. A progressive increase in the distensibility of the eyes was noted at 12 hours to four days when compared to eyes which were tested in situ 30 minutes after death.

Development of stromal collagen patterns. Alfred J. Coulombre, Ph.D., and Jane L. Coulombre, B.S., Department of Anatomy, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut.

The arrangement of stromal collagen is an important factor in the transparency of the cornea. This study deals with the nature of this pattern in the chick embryo, and the manner in which it develops. Collagen fibers are present in the developing corneal stroma from very early stages. It is not until the eighth or ninth day of incubation, however, that collagen fibers are deposited in a geometric array. At this time collagen fibrils and fibroblasts tend to allign in parallel fashion. Shortly thereafter a second rank of fibers and cells is observed with its long axis at right angles to the first rank. In this manner a rectangular gridwork of fibers and cells is formed which lies parallel to the surface of the cornea. Similar layers are subsequently formed. The axes of any one layer are not in register with those of adjoining layers.

Freshly excised corneas of different ages were studied with polarized light. The Maltese cross pattern, which is typical of the adult cornea, appears during development, with the fiber patterns described above.

On the basis of this and other studies it is postulated that: (a) mechanical factors acting on the developing cornea are responsible for the pattern of deposition of collagenous bundles in the stroma, and (b) that the manner of deposition of collagen in the cornea assures its development as a segment of a sphere.

The vitreous cells and their role in the formation of the vitreous body. Endre A. Balazs, M.D., Lars Sundblad, M.D.,

and Laszlo Z. J. Toth, Ph.D., Retina Foundation, Department of Ophthalmology, Massachusetts Eye and Ear Infirmary and Harvard Medical School, 30 Chambers Street, Boston.

It has been reported that the concentration of hyaluronic acid and certain proteins in the adult cattle vitreous body is highest in the cortical layer, next to the retina, and gradually decreases toward the center, reaching the lowest value in the anterior segments next to the lens (Balazs, E. A.: Structure of the vitreous gel. Acta XVII Conc. Ophth. **2**:1019, 1955). Later it was reported that this cortical layer contains a special cell, which is embedded in that portion of the vitreous gel containing the highest concentration of hyaluronic acid (Szirmai, J. A., and Balazs, E. A.: Studies on the structure of the vitreous body: III. Cells in the cortical layer. Arch. Ophth., in press). It was found that the vitreous cells are already present in the cortical layer of the vitreous body of four- to five-month-old cattle embryos and that their total number increases with the growth of the eyeball. In order to determine whether or not these cells participate in the formation of hyaluronic acid in the eye, in vitro experiments were carried out to study the incorporation of C^{14}-labeled glucose into the hyaluronic acid molecule in the presence of living cells and cell homogenates. The results indicate that these cells can form high-molecular (nondialyzable) hyaluronic acid in vitro.

The relation of the adrenal gland to experimental production of exophthalmos. George K. Smelser, Ph.D., and Victoria Ozanics, M.S., Department of Ophthalmology Research, College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32.

Recent studies have shown that the degree of the exophthalmos which can be produced by the injection of the pituitary extracts in guinea pigs is greatly increased by adrenal steroid hormones. This suggests the possibility that the orbital changes causing exophthalmos depend on the presence of adrenal steroids. To test this hypothesis attempts were made to produce exophthalmos in guinea pigs in the complete absence of these steroid hormones. The adrenal glands were removed from large female animals in two operations. The ovaries were also removed, at the time of adrenalectomy, so that no ovarian steroids were present. In addition to adrenalectomy and ovariectomy, thyroidectomy was done as in earlier studies. Maintenance of the thyroidectomized, ovariectomized and adrenalectomized guinea pigs, especially those receiving pituitary extract injections, proved exceedingly difficult, but was finally accomplished by the frequent oral administration of glucose and sodium chloride, and housing the animals in heated draft-free cages. The pituitary preparation used was prepared by Organon, Inc. and contained considerable thyrotropic hormone.

It was demonstrated that exophthalmos could be produced in the total absence of steroid hormones from the adrenal and ovary. The exophthalmos in these cases was qualitatively similar to that of guinea pigs with intact ovarian and adrenal glands, but was much less marked. The adrenal steroids, therefore, although they play an important role in the severity of experimental exophthalmos, are not "permissive" agents. That is, the production of exophthalmos and the accompanying pathology of the orbital connective tissue does not depend on the presence of these steroid hormones.

The mast cells in the uveal tract of the eye and changes induced by hormones. Godfred Larsen, M.D., Laboratory for Connective Tissue Research, Univer-

sity Institute of Medical Anatomy, Copenhagen, Denmark. Mailing address: Department of Ophthalmology Research, College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32.

Mast cells, one of the cell types of all connective tissue, are present in considerable but variable amounts in the uveal tract of the eye, the number, however, besides individual variations, depending on the species of animals.

Rabbits, rats, mice and guinea pigs were used in the experiments. The mast cells were found most numerous in the uvea of the guinea pig, and in decreasing numbers in the following order in rats, mice and rabbits. In the uvea itself by far most mast cells were present in the choroid, less numerous in the ciliary body and very few to none in the iris, always in close relation to the vessels. Both whole eyes and pieces of eyes were used, fixed in four-percent lead subacetate or 0.4-percent 5-acridine hydrochloride dissolved in 50-percent alcohol. Nearly all specimens were stained with toluidine blue, hematoxylin-eosin and Hotchkiss' stain.

By treatment the mast cells changed their appearance. Cortisone decreased the number of mast cells and the cytoplasm showed signs of degranulation and lumping of the granules with formation of vacuoles. Thyroidectomy and especially thyroidectomy combined with treatment with thyrotrophine increased the amount of mast cells and the cytoplasm became more or less saturated with granules. Following treatment with thyroxine the mast cells were less numerous and smaller, with sparse granulation of cytoplasm. C-avitaminosis provoked heavy degranulation of the mast cells.

The function of mast cells in the uvea is presumably the same as in connective tissue elsewhere, namely secretion of mucopolysaccharides. Because of the complicated structure of the eye, the mast cells in the uvea may be of importance for the fluids of the eye, where mucopolysaccharides are also found.

Heterologous immunologic studies with lens. Patricia L. FitzGerald, B.A., and Seymour P. Halbert, M.D., Department of Ophthalmology Research, College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32.

Earlier studies have shown that rabbits can be readily immunized with homologous rabbit lens with the aid of Freund's adjuvant as a vehicle. Such sera have revealed at least five distinct antigen-antibody systems with agar precipitin techniques. Cross reactions of these sera with lenses from a wide variety of vertebrates were shown to be due to antigenic similarity of from one to all five of the components visualized.

Heterologous antisera have now been prepared in rabbits to lenses of several other species, human cataract, fish (menhaden), frog (rana pipiens) and squid. All of these antisera show from at least five to seven antigen-antibody systems with their immunizing lens. The antisquid lens antisera failed to show any cross reactions with any of the vertebrate lenses tested and revealed up to five antigens with the squid lens. All of the vertebrate antilens sera failed to show any precipitin bands with the squid lens. These findings indicate that the evolutionary development of lens proteins may have taken place along two different lines. The number of cross reactions of the vertebrate lens antisera with lenses of other species essentially agree with the cross reactions found with homologous rabbit antilens sera.

Effect of irradiation on intraocular antigen-antibody reactions. S. James Bullington, M.D., and Arthur F. Howe,

Ph.D., Retina Foundation, Department of Ophthalmology, Massachusetts Eye and Ear Infirmary and Harvard Medical School, Boston. Retina Foundation, 30 Chambers Street, Boston 14.

The effect of irradiation on the intraocular Arthus reaction has been studied. The passive intraocular Arthus reaction was previously standardized in normal rabbits. In rabbits given doses of total-body irradiation (> 400 r) sufficient to lower the polymorphonuclear neutrophil count to fewer than 2,000, the passive intraocular Arthus reaction was found to be markedly inhibited. With total-body irradiation doses of less than 400 r, there was little inhibition of the passive reaction and no noticeable effect on the intraocular Arthus reaction in actively sensitized animals. Clinical and histologic studies of the eye were made in rabbits in which passive Arthus' reactions had been produced repeatedly. It was found that in some rabbits in which the Arthus reaction was produced in one eye, a simultaneous reaction was observed in the unmanipulated eye.

Retinal detachment has been observed in some eyes which received intravitreal antigen injections followed by an antigen-antibody reaction (antibody injected intravenously). No retinal detachment has been observed in eyes which received only intravitreal saline injections.

Two early wound reactions in rat corneas. Virginia Weimar, Ph.D., Department of Ophthalmology Research, College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32.

Studies with neutral red vital staining have revealed an early wound reaction in the stromal cells of rat corneas. Under the conditions of these experiments the corneal stromal cells in unwounded eyes were negative to neutral red. Within the first 12 postoperative hours *all* of the corneal stromal cells became transformed to macrophagelike cells which phagocytosed large quantities of the dye. The reaction became still more intense by 24 hours after injury. The fibroblastlike cells of the wound edge also took up the dye intensively.

Thirty-six hours after wounding, however, a new phenomenon occurred. Some of the fibroblasts at the wound edge took up no neutral red at all, although a large number of them did. The cells away from the wound edge continued to take up the dye intensively.

By 60 hours after wounding the fibroblasts of the wound edge were no longer able to phagocytose the neutral red. The cells away from the wound edge continued to take up the dye for as long as 96 hours (the longest time period studied).

The results were interpreted as:

1. Within 12 hours after wounding an unknown injury factor released by the wound reaction causes the transformation of all the corneal stromal cells to macrophagelike cells. These continue to behave like macrophages until at least 96 hours after wounding.

2. At the wound edge a second unknown factor, released by the wound reaction, transforms macrophages, derived from either corneal stromal cells or monocytes, into fibroblasts.

Some hypothetic wound factors and their relation to these phenomena were discussed.

Experimental ocular carcinogenesis: I: The effect of intraocular methyl cholanthrene implantation on the mouse eye. Arnall Patz, M.D., Lydia Wulffe, M.S., and Sarah Rogers, Wilmer Institute, The Johns Hopkins Hospital, Baltimore 5, Maryland.

Methyl cholanthrene, an extensively investigated carcinogenic hydrocarbon, was

introduced into the mouse eye in these studies.

Minute pellets of fused cholanthrene crystals or the fine powdered crystals themselves, were introduced by a No. 25 needle in the following experiments: (1) directly into the anterior chamber; (2) into the vitreous; (3) directly into the retina. The animals were all inbred cancer strain mice, varying from five days to three months of age. Approximately 250 animals were operated and the animals were killed after a follow-up of from one to 18 months.

The most interesting observation from these experiments was that the surface epithelium near the sight of needle puncture proliferated into the wound to invade the anterior or posterior chamber of the eye. This occurred in approximately one fourth of the operated animals (62 eyes). Serial sections demonstrated the continuity through the wound of surface and ingrown epithelium. In several instances the ingrown surface epithelium proliferated markedly showing precancerous changes. In one instance a squamous-cell carcinoma developed from the ingrown epithelium.

In six eyes the retinal pigment epithelium showed marked proliferation. Several mesodermal tumors (sarcomas) occurred. However, the exact origin of these tumors could not be determined. No true neoplastic changes were detected in the retinas.

Distribution of sulfhydryl groups and proteins in calf and bovine lens. Jin H. Kinoshita, Ph.D., and Lorenzo Merola, B.S., Harvard Medical School and Massachusetts Eye and Ear Infirmary, Howe Laboratory, 243 Charles Street, Boston 14.

In the course of studying the effect of the aging process on the lens an attempt was made to relate the distribution of the sulfhydryl (-SH) groups to the different types of protein normally found in lens. To gain insight on what changes may occur as the lens fibers become older, chemical analyses and electrophoretic studies were made on the cortex and nucleus of young and old lenses. In these studies the lens from the young animals were obtained from calves and the older lens were from cattle.

It is apparent from the results with cattle lens that the cortex is rich in glutathione. In the cortex of cattle lens the concentration of this tripeptide on a dry weight basis is almost five times that of the nucleus. The interesting feature of this distribution is that although there is a high concentration of glutathione, the protein sulfhydryl (P-SH) content is lower in the cortex than in the nucleus. In fact there seems to be a close reciprocal relationship between the cortex and nucleus in regard to the distribution of glutathione and P-SH. These findings lead one to speculate that as the cortical fibers age and become part of the nucleus, glutathione disappears and, in its place, an equivalent amount of P-SH is formed. This may be the result of either the -SH of glutathione actually being incorporated into the protein molecule or being involved in the reduction of a disulfide bond of the proteins. It is interesting to note that along with this predicted transformation another protein appears in the nucleus. Electrophoretically the cortex is made up of two proteins: alpha-crystallin, the -SH poor protein and beta-crystallin, the -SH rich protein. In the nucleus electrophoretic patterns not only show two proteins having mobilities corresponding to the alpha and beta crystallins of the cortex, but also a third protein, the gamma-crystallin.

In comparing the young with the old lens it appears that the P-SH of calf lens is much higher in both the cortex and nucleus. Electrophoretic studies seem to furnish an explanation for this difference.

In calf lens there is only a small amount of alpha-crystallin, the -SH poor protein. The majority of the calf lens proteins in the cortex and nucleus is made up of beta and gamma crystallins. The gamma-crystallin is present in strikingly high concentrations in calf lens. Again in the cortex and nucleus of calf lens a reciprocal relationship in the distribution of glutathione and P-SH is observed.

Quantitative histochemical changes in the development of rat lens and cornea. Robert E. Kuhlman, M.D., and Robert A. Resnik, Ph.D., Ophthalmology Branch, NINDB, National Institutes of Health, Bethesda 4, Maryland.

The activity of the following enzymes was determined in cornea and lens tissue at various stages of development of the rat's eye: hexokinase, aldolase, lactic dehydrogenase, isocitric dehydrogenase, glucose-6-phosphate dehydrogenase, glutamic dehydrogenase, and glutathione reductase. Each enzyme was assayed in frozen dried sections of corneal epithelium, corneal stroma, lens capsule with cortical epithelium, lens cortex, and lens nucleus of new born, 10-day, 20-day, and adult (100-day) rats. Corneal epithelium had the highest over-all activity. Lactic dehydrogenase was the most active system. The enzymatic activity of corneal stroma and lens nucleus decreased with growth while that of the corneal and lens epithelium either increased or remained relatively constant.

The findings of Kinoshita, indicating a high activity of the direct oxidative pathway, have been confirmed. In addition, isocitric dehydrogenase was found to have the same magnitude as does glucose-6-phosphate dehydrogenase.

Glutamic dehydrogenase activity is very low.

Hexokinase and aldolase appear to be limiting systems in all the tissues. The capacities of both these systems remain low throughout development.

Oxidation of soluble lens proteins by heavy metals in presence of phosphate. Zacharias Dische, M.D., Joy Elliott, B.S., and Ginevra Zelmenis, B.A., Department of Ophthalmology Research, College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York 32.

It has been reported by Croisy and Dische that the cysteine of soluble proteins of rat lenses is oxidized by 0.1 to 1.0 M phosphate. As oxidation of reducing substances by phosphate is known to be a catalysis by traces of heavy metals present in phosphate solutions, experiments were carried out to determine the respective roles of heavy metal and phosphate in the oxidation of soluble lens proteins. Proteins of lenses of rats and cattle prepared by centrifugation of 1:10 homogenates of lenses were used. Cu^{++} in $4 \cdot 10^{-6}$ M to 10^{-5} M, Fe^{++} in $4 \cdot 10^{-5}$ to $2 \cdot 10^{-4}$ were tested in presence of phosphate in concentrations ranging from 1/3000 to 0.3 M. Cu^{++} at $2 \cdot 10^{-5}$ M in 0.3 M phosphate oxidized twice as much cysteine in 2½ hours at 37°C. at pH 7.4, then phosphate alone. In 0.03 M phosphate, the oxidation by Cu was much smaller and was barely significant in 1/3000 M phosphate. This indicates that a phosphate complex of Cu is the catalyzing agent. Fe^{++} at $2 \cdot 10^{-4}$ M was less effective than Cu at $2 \cdot 10^{-5}$, while the oxidation by Fe^{+++} at $2 \cdot 10^{-4}$ was barely significant. The cysteine which disappeared by oxidation was recovered as cystine. When lens proteins were pre-incubated with phosphate alone or phosphate and Cu for two hours at 37°C. and then incubated with $K_3Fe(Cn)_6$, the reduction of the latter was strongly decreased as compared with the simultaneous reduction by nonpreincubated proteins; Cu combined with phosphate, however, did not decrease the reduction

more than phosphate alone, which indicates that Cu at $2 \cdot 10^{-5}$ M increases the availability of reducing groups of soluble lens proteins. The influence of pH on these oxidations has been examined. As this oxidation process is taking place at concentrations of Cu^{++} and phosphate which can be regarded as physiologic, it could be responsible for the continuous accumulation of cystine in ocular lenses during aging and cataract formation.

Hexokinase activity of rabbit lens: Effect of age. Harry Green, Ph.D., and Shirley A. Solomon, B.S. (With technical assistance of Caroline A. Hamblin, M.S.), Research Department, Wills Eye Hospital, 1601 Spring Garden Street, Philadelphia 30.

Homogenates of lenses prepared from three to four-week and three-year-old rabbits were used to study the effect of age upon the hexokinase activity in the crystalline lens. With both age groups, under optimal conditions of pH and concentration of glucose, the enzymatic activity, as indicated by the rate of disappearance of glucose, was linear with time, at least up to 30 minutes, under the given experimental conditions. Characterization of the enzymes from the two age-different lenses; for example, pH optimum, optimal substrate concentration, Michaelis-Menten constant indicate that with age the biochemical characteristics of the hexokinase are unaffected. Furthermore, while the total enzyme activity of the lens did not change appreciably with age, the concentration in the older lens was markedly lower than in the younger lens. The distribution of hexokinase between the nucleus, cortex and capsule-epithelium was also studied. The results will be discussed in terms of the growth characteristics of the lens.

The effect of glutathione in the formaldehyde oxidation of the retina. Jin H. Kinoshita, Ph.D., Harvard Medical School and Massachusetts Eye and Ear Infirmary, Howe Laboratory, 243 Charles Street, Boston 14.

It is of especial interest to study the metabolism of formaldehyde in the retina because of the possibility of this compound being the toxic agent in methanol poisoning. Potts and Johnson have tested the toxic effects of methanol, formaldehyde and formic acid and found that formaldehyde, the first metabolite of methanol oxidation, was the most effective inhibitor of many of the retinal enzymes. Therefore, it is implied that in those individuals who are susceptible to methanol poisoning formaldehyde accumulates in the retina either because more of it is available or it is not metabolized rapidly enough. Heretofore, it was thought that the normal course of formaldehyde metabolism was its oxidation to formic acid catalyzed by a nonspecific enzyme, acetaldehyde dehydrogenase. It has recently been shown that in liver a specific enzyme, formaldehyde dehydrogenase, is present which oxidizes formaldehyde and requires glutathione as a co-factor. In the current report evidence will be presented which indicates that this specific dehydrogenase also participates in the formaldehyde oxidation of the retina.

In the dialyzed extracts of bovine retina, it has been possible to demonstrate the oxidation of formaldehyde and acetaldehyde by following the reduction of DPN spectrophotometrically. In the retinal extracts the oxidation of acetaldehyde occurred at a much more rapid rate than that of formaldehyde. However, when glutathione was added to the reaction mixture, the formaldehyde oxidation was stimulated to such an extent that the rate of its oxidation was faster than that of acetaldehyde. The presence of glutathione, although it had a marked stimulatory effect on the formaldehyde oxida-

tion, did not alter the rate of oxidation of acetaldehyde. It has been shown that the end-product of the formaldehyde oxidation is formic acid. In some preparations of the retinal extracts in which all the glutathione was removed no oxidation of formaldehyde was observed until a trace of glutathione was added to the reaction vessels. The requirement of glutathione in the oxidation of formaldehyde has been demonstrated in the extracts from cattle, rabbit, and human retinas.

Assuming that in methanol poisoning formaldehyde is the toxic agent, it is difficult to say at this time without more evidence whether a deficiency of this enzyme or the lack of available glutathione may contribute to the symptoms observed.

Electron microscope study on the lens epithelium. Theodor Wanko, M.D., and Mary Ann Gavin, M.A., Ophthalmology Branch, NINDB, National Institutes of Health, Bethesda 14, Maryland.

The epithelium of the lens has been studied on specimens from adult normal animals of various vertebrate species (rat, guinea pig, rabbit). The general cytologic pattern and the morphologic relationship of epithelial cells to lens capsule and lens fibers are similar in these species. The lens fibers are in juxtaposition with the epithelial layer, separated by a less opaque space between two dense cellular membranes. A boundary between epithelium and lens capsule consists only of the anterior epithelial cell membrane. In the region near the capsule the epithelial cells interdigitate frequently, whereas the cell membranes meet in a fairly regular fashion in the zone near the lens fibers. Cell nuclei form round or ovoid rather homogenous profiles, outlined by a double membrane. They are more opaque than the surrounding cytoplasm and their periphery sometimes appears indented. Nucleoli can be distinguished within the body of the nucleus by their greater density. In all types of lens epithelial cells four major cytoplasmic constituents can be differentiated: mitochondria, endoplasmic reticulum of the granular type, endoplasmic reticulum of the agranular type, an elongated, very small vesicular component and a structure suggestive of Golgi equivalent. It appears that the quantitative relationship between some of these cytoplasmic constituents varies in different regions of the lens epithelium.

A clinicopathologic and histochemical study of calcium oxalate crystallization within ocular tissues. Lorenz E. Zimmerman, M.D., and Frank B. Johnson, M.D., Central Laboratory for Pathologic Anatomy and Research, Veterans Administration, Armed Forces Institute of Pathology, Washington 25, D.C.

For a number of years, pathologists at the Armed Forces Institute of Pathology had recognized the presence of nonstaining birefringent crystalline deposits occurring within certain ocular tissues. These cases were recorded in the Registry of Ophthalmic Pathology under the tentative heading of "protein crystals," but the chemical nature of the crystals remained unknown until the present investigators became curious about them early in 1954. A new histochemical procedure devised by one of us (F.B.J.: J. Histochem. & Cytochem., **4**:404-405, 1956) led to their being identified as crystals of calcium oxalate. The occurrence of these deposits was recognized in two main types of cases, one of which was phacolytic glaucoma. Hence brief mention was made of their presence in the sclerotic nuclei of morgagnian cataracts in the paper on phacolytic glaucoma by Flocks, Littwin, and Zimmerman (Arch. Ophth., **54**: 37-55, 1955).

Independently and without knowledge of our interest in these crystals, Dr.

David G. Cogan and his associates at the Howe Laboratory in Boston discovered identical crystals in an eye obtained from a 13-year-old boy who had a six-year history of uveitis which progressed to retinal detachment and secondary glaucoma. This case was presented at the April, 1956, meeting of the Ophthalmic Pathology Club at which time the results of our own studies were also reported. Subsequently Cogan and coworkers observed these crystals in other cases and by means of chemical and X-ray diffraction studies identified them in at least two instances as consisting predominantly of monohydrated calcium oxalate with a lesser amount of calcium phosphate (Cogan, D. G., et al.: Calcium oxalate and calcium phosphate crystals in detached retinas, in press).

The crystals as they are found in ordinary tissue sections are characterized by their insolubility in aqueous formalin, alcohol, and such fat solvents as are used in the preparation of celloidin and paraffin sections; by their lack of significant staining with hematoxylin and eosin; and most significantly by the colorful birefringence which they exhibit with polarized light. The possibility that they might represent calcium oxalate was first suspected because of their very characteristic appearance and their similarity to crystals found in cases of oxalosis and ethylene glycol poisoning and to the calcium oxalate crystals described in the thyroid gland by Richter and McCarty (Am. J. Path., **30**: 545-554, 1954). The previously cited histochemical test devised by Johnson for the identification of calcium oxalate in tissue sections is based upon its being converted into calcium carbonate when heated at 450°C. for 20 minutes. Then while examining the incinerated tissue sections microscopically, a drop of dilute hydrochloric acid is allowed to cross the section. When the acid makes contact with the calcium carbonate, the latter promptly disintegrates liberating many gas bubbles (carbon dioxide). The two-step reaction involved is:

$$(1)\ CaC_2O_4 \xrightarrow{\text{heat}} CaCO_3 + CO$$

$$(2)\ CaCO_3 \xrightarrow{\text{acid}} CaO + CO_2$$

Control sections treated with acid before microincineration reveal the calcium oxalate crystals to dissolve but no gas is released.

Clinically and histopathologically the cases in which these calcium oxalate crystals have been found fall into two main groups. One consists of aged patients with morgagnian cataracts in which cases the crystals are found in the sclerotic lens nucleus. The other group represents cases of long-standing retinal detachment, usually the result of a penetrating injury sustained in childhood. In these cases the crystals are found in the outer layers of the degenerated retina and sometimes free in the adjacent subretinal exudate.

The metabolic significance of calcium oxalate crystals in these cases is unknown.

Further histochemical studies of acid mucopolysaccharides in the intraocular tissues. Lorenz E. Zimmerman, M.D., Central Laboratory for Pathologic Anatomy and Research, Veterans Administration, Armed Forces Institute of Pathology, Washington 25, D.C.

Previous studies based on human eyes enucleated surgically, fixed in aqueous formalin, imbedded in paraffin, stained by the colloidal iron and alcian blue technics, and treated with commercially available bovine testicular hyaluronidase have made possible the following observations: (a) A mucopolysaccharide sensitive to bovine testicular hyaluronidase normally fills the intertrabecular spaces between

the anterior chamber and the canal of Schlemm; (b) the vitreous contains similarly sensitive acid mucopolysaccharide; (c) a different acid mucopolysaccharide which resists bovine testicular hyaluronidase forms a ground substance in which the outer segments of the visual cells are imbedded; (d) a similarly resistant mucoid is formed by retinoblastomas; (e) hyaluronidase-sensitive acid mucopolysaccharide replaces the parenchyma of the optic nerve in cavernous optic atrophy and fills the spaces formed in microcystic degeneration of the peripheral retina.

A limited number of observations based on eyes removed at autopsy suggested that there is a rapid deterioration of acid mucopolysaccharides as a result of agonal or postmortem changes so that they no longer give satisfactory staining reactions. It is therefore very difficult to obtain satisfactory material for histochemical characterization of the completely normal human eye.

Further studies have been undertaken along the following avenues: (a) The normal rhesus monkey eye fixed by a variety of technics has been used as a "substitute" for human material in an effort to obtain more information about the normal distribution of acid mucopolysaccharides; (b) a relatively pure streptococcal hyaluronidase has been obtained through the kindness of Dr. Emily W. Emmart of the National Institutes of Health, in order that the effects of this enzyme could be compared with those of the bovine testicular preparation since it is believed to be the more specific for hyaluronic acid; (c) the development of visual cells after birth is being studied and compared with the formation of the mucopolysaccharide ground substance in which the outer limbs of these cells are imbedded, using both pigmented and nonpigmented mice and rabbits. The genetically blind C_3H mouse is also being used in these studies which represent attempts to determine the source of this particular acid mucopolysaccharide.

Progress made along these three main avenues of histochemical investigation will be reported.

Cytomegalic inclusion disease: Isolation of virus from anterior chamber of eye. Robert P. Burns, M.D., College of Physicians and Surgeons, 635 West 165th Street, New York 32.

Cytomegalic inclusion disease is a widespread, long known and little understood disease from which a virus has recently been isolated. This virus produces cytopathogenic changes in tissue culture comparable to the histopathologic changes.

The syndrome is frequently seen in newborn infants and closely simulates toxoplasmosis. A case was observed in which chorioretinitis occurred. The virus was isolated from the urine and typical inclusion bodies were seen in urinary sediment.

An attempt was made to define the etiology of the chorioretinal disease by anterior chamber puncture. No inclusion bearing cells were found in the aqueous, and one attempt to culture virus failed. On a second attempt, cytopathogenic changes with production of typical inclusion bodies were found in tissue cultures of human embryonic fibroblasts maintained in Eagle's medium with 10-percent horse serum. Serial reproduction of these cytopathogenic changes was accomplished.

It is felt that isolation of a virus from the anterior chamber tends to confirm the pathologic finding of inclusion bodies in enucleated eyes and establishes this etiologic agent as one cause of uveitis.

The effect of nystatin and amphotericin B on experimental corneal infections with Candida albicans. Theodore W. Sery, Ph.D., and Jerome A. Montana,

M.D., University of Pennsylvania Graduate School of Medicine, Wills Eye Hospital, Research Department, 1601 Spring Garden Street, Philadelphia.

Nystatin and Amphotericin B, two of a new group of fungistatic antibiotics, were tested in rabbits to determine their ocular toxicity, ocular penetration and therapeutic effect against C. albicans infections of the rabbit cornea. Amphotericin B which could be dissolved in five-percent aqueous glucose solution to the extent of 5.0 mg./ml. showed no toxic effects to ocular tissues when it was applied topically, subconjunctivally (0.3 ml.) and intravenously (1.0 ml.). The intravenous inoculation, however, had to be administered slowly over a period of five minutes otherwise an occasional death would result. Nystatin, which is not water soluble, was found to be injurious to the eye when given subconjunctivally in saline suspension (4,000 units/ml.) in amounts of 2,000 units or more; topical application of the saline suspension or a propylene glycol solution in an ointment base was nontoxic in concentrations as high as 100,000 units/ml.

The antibiotics were not found in detectable amounts in the aqueous humor after administration by local application on the intact or abraded corneal epithelium or by intravenous or subconjunctival injection (Nystatin, locally 100,000 units/ml. subconjunctivally, 800 units; Amphotericin B, locally 5.0 mg./ml., subconjunctivally 1.0 mg., intravenously 5.0 mg.).

Corneal infections were difficult to produce with C. albicans alone even when injected intracorneally in high doses. However, an intracorneal injection of Prednisolone (30-60 μg.) preceding an intracorneal injection of C. albicans cells (10^5 cells in 0.01 ml.) gave a reproducible lesion that led to penetration of the globe within a week. When the period between the injection of steroid hormone and C. albicans was increased it was found that the infection enhancing property of the drug was effective for at least two weeks.

Positive inhibition of C. albicans infections of the cornea could be effected with either drug if treated early enough and with the proper route of administration. Nystatin was effective within 24 hours by topical application and Amphotericin B was effective within 48 hours by the topical and intravenous routes of administration. Neither drug was able to prevent infection when given subconjunctivally. The data suggest that Amphotericin B is able to penetrate the corneal stroma from the limbal vessels as well as through the intact epithelium. The failure of the subconjunctival injection of antibiotics to alter the course of corneal infection infers that the subconjunctival and scleral tissues offer a relatively impermeable barrier to those antibiotics or at least that penetration did not take place before the drug was removed from this site.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM*

77th Annual Congress

April 11-13, 1957

MR. J. J. HEALY, *President*

INTRAOCULAR CIRCULATION AND HYPERTENSION

MR. J. J. HEALY, in his presidential address, described intraocular circulation in arteriosclerosis and hypertension. He discussed the etiologic and pathogenic theories of hypertension, which were largely based on the evidence of experimentally produced and secondary hypertension, and he noted that although physiopathologic research had made some progress, no definite results had yet emerged. Arteriosclerosis might be present as a senile regression in the retinal and choroidal vessels, and essential hypertension might supervene or arise as a separate entity. Mr. Healy analyzed the changes in 200 outpatients' fundi and assessed them with special reference to angiospasm, arteriolosclerosis, and arteriolonecrosis; and he made a plea for greater liaison between physician, ophthalmologist, and pathologist.

SIMPLE GLAUCOMA: THE BOWMAN LECTURE

SIR STEWART DUKE-ELDER discussed the mechanism of the formation of the aqueous humor in the light of recent biochemical and pathologic research, with particular reference to the extensive work carried out under his direction at the Institute of Ophthalmology. He then turned to the variations in intraocular pressure produced especially by peripheral and central nervous stimulation, and he indicated the factors responsible for the instability and range of variation of the intraocular pressure characteristic of the glaucomatous eye. He traced the development of the initial functional and consequent organic changes with their terminal ischemic effects upon the structures of the anterior and posterior segments of the eye and the optic nerve, and he examined their respective roles in the development of clinical simple glaucoma. Glaucoma should not be regarded purely as an ocular disease, but rather as a local manifestation of a more general disorder. Advances in the scientific aspects had not been accompanied by similar advances in either medical or surgical treatment, which had made little progress during the past 20 years.

CONGENITAL CATARACT

MR. J. H. DOGGART who introduced a symposium on this subject, said that, although some slight opacity was visible in most human lenses, even among the newborn, most of these partial cataracts were harmless and stationary. Nevertheless, they could not be dismissed as insignificant because (a) they might be misinterpreted and (b) they had supplied clues for embryologic investigation. Differentiation of the various congenital cataracts from other kinds of opacity was impossible without careful study of the living lens.

MR. A. B. NUTT emphasized that the results of surgery for congenital cataract were disappointing and that the prognosis must therefore be guarded. Having classified the cases requiring surgical treatment, he gave a brief resume of the operations commonly employed, their complications, and the methods adopted to avoid them. He thought that the poor functional results were in many cases due to associated congenital anomalies.

* Reprinted from *The Lancet*, April 27, 1957, pp. 868-869.

Ocular aspects of diabetes

Prof. G. I. Scott classified the retinal changes of diabetes and discussed their possible etiology. This form of retinopathy could occur in the absence of any evidence of hypertension or arteriosclerosis and its incidence seemed to depend primarily upon the length of time that the patient had suffered from the disease.

Dr. J. D. N. Nabarro expressed some alarm at the increasing incidence of serious visual defects due to diabetic ocular disease. The speed with which vision deteriorated in patients with established retinitis was extremely variable and it was uncertain whether it could be influenced by treatment. Strict diabetic control was essential, but there was no evidence that vitamins or sex hormones had any effect. Hypophysectomy and pituitary destruction were being undertaken in a few centers, but their value was still uncertain.

Dr. Norman Ashton dealt with the experimental side of diabetic vascular disease, with special reference to its ocular and renal manifestations. He attempted to assess the bearing of experimental findings on the relationship between diabetic retinopathy and glomerulosclerosis.

Retinal detachment

Mr. P. McG. Moffatt and Mr. C. Dee Shapland discussed the role of intravitreal vitreous injection in retinal detachment and described the technique for obtaining the donor vitreous and its subsequent injection under pressure into the recipient eye. Mr. Shapland analyzed the results obtained in a small series of his own cases and those of Mr. Moffatt.

Mr. George Black criticized present-day operations in regard to their effect upon the sclera, such as local necrosis, injury to nerves and blood-vessels, and upon anterior structures. The effects of diathermy treatment were variable when given through the full thickness of the sclera, and Mr. Black described his own method of scleral trap-door access to the choroid and its application.

Ptosis operation

Mr. H. B. Stallard advocated the anterior surgical approach to the levator palpebræ superioris, which exposes it in its normal relationship to other structures, reveals any unusual attachment of this muscle to the orbital fascia, allows the separation of any fascial connection with the superior-oblique tendon sheath, and permits a greater length of the muscle to be resected and more accurate suturing of the resected muscle to the tarsal plate than other methods of exposure. He described a new lidguard for use in this operation and illustrated the technique with a colored film.

Phacolytic glaucoma

Dr. P. Schofield gave an account of the association of glaucoma with hypermature cataract. Although recognized since 1900, this condition had only recently been defined as a clear-cut pathologic entity and named "phacolytic glaucoma." From the histologic examination of three cases, Dr. Schofield described the pathology and pathogenesis of the condition.

Venous obstruction at the disc in chronic glaucoma

Mr. J. H. Dobree described macroscopic lesions on glaucomatous optic discs, including central-vein thrombosis, venous coils, anastomotic vessels and hemorrhages at the disc margin. These lesions had been seen in a series of 200 cases of chronic glaucoma. He discussed the relationship of these vascular changes to other signs of glaucoma such as the prevailing level of tension, cupping, atrophy, and field defects. In a control series without tension but with gross arteriosclerotic changes in the vessels, the disc appearances were quite different. The main factor determining the onset of the venous lesion was probably the glaucomatous cupping.

Foreign-body detector

Mr. M. J. Roper-Hall described a new electronic instrument which could detect small metallic particles, both magnetic and nonmagnetic, and assess their chemical and physical properties. Two sizes of probe had been made, one 0.25 in. and the other 0.5 in., the latter being sensitive to very small metallic fragments at a distance of 15 mm. The prototype instrument had already been used to confirm the presence of intraocular foreign bodies originally detected by X-ray examination and localization, and it had also been used to identify metallic fragments seen on radiographs as magnetic or nonmagnetic. The instrument was insensitive to other metal bodies such as dental fillings and the operating table at distances greater than three cm. The sensitivity of the probes increased with their size, but so did the range within which other metal bodies affected them (the 0.5 in. probe was unaffected by metal bodies at a distance greater than four to five cm.). Mr. Roper-Hall said that the instrument was still being developed and that its uses were being extended from ocular to general surgery.

Uveitis in childhood

Mr. Joseph Minton gave an account of three children with severe uveitis, in one of whom the full clinical picture of Still's disease developed some time after the original uveitis. Cortisone and corticotrophin, and more particularly the new derivatives, prednisone and prednisolone, were valuable in treatment. A child who had responded indifferently to cortisone was much improved by prednisolone.

Ocular onchocerciasis

Dr. F. C. Rodger described an extensive field survey and clinical and experimental work undertaken to determine the types of onchocerciasis in which ocular lesions were most likely. To estimate the density of infection he calculated an index which related the degree of infestation and its anatomic distribution over the body with the likelihood of ocular onchocerciasis. He found cases in which infectivity was maximum by this standard of examination, and the eyes had actually been invaded, yet there were no ocular lesions. This he attributed to an immunity which developed because of infestation in other parts of the body. Experimental work with animals has shown conclusively that it was the dead parasite which led to an inflammatory reaction, and nearly all the manifestations of human onchocerciasis were reproduced in the rabbit. Dr. Rodger found in some cases with posterior-segment lesions, particularly choroidoretinal degeneration, that there was a low individual density figure, and the damage to the tissues might be due to a toxin secreted by the adult worms.

Conjunctivitis at Moorfields in 1956

Mr. Barrie R. Jones had investigated a proportion of the cases presenting at Moorfields Hospital with conjunctivitis during 1956, in order to establish the etiology of the condition. The particular aim was to evaluate the role of various viruses in the production of conjunctivitis and keratoconjunctivitis. A few infections were due to some of the well-recognized bacterial infections; several cases were associated with infection by adenoviruses; and in others there was evidence of infection by the herpes-simplex virus. Among other infections seen in association with conjunctivitis were molluscum contagiosum, vaccinia, influenza, and cat-scratch fever.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

December 10, 1956

Dr. Max Chamlin, *President*

Recent advances in medical therapy of glaucoma

Dr. Irving H. Leopold delivered the 10th annual Mark J. Schoenberg Memorial Lecture, under the auspices of the New York

Society for Clinical Ophthalmology and the National Society for the Prevention of Blindness.

The medical methods of lowering intraocular pressure in primary glaucoma consist of: Parasympathomimetic agents (pilocarpine), sympathomimetic agents (adrenalin), hormonal agents (pituitary extracts), osmotherapeutic agents (Sorbitol), enzymatic agents (Diamox), antihypertensive and tranquilizing agents (hexomethonium, Ansolysen, thorazin), diuretics (Thiomerin), anesthetics (pentothal, procaine). These compounds can be classified on the basis of their mode of action, that is, (1) those which reduce the resistance to outflow of intraocular fluid (miotics) and (2) those which reduce the formation of intraocular fluids (Diamox).

The onset of medical therapy of glaucoma really began with the use of eserine and pilocarpine in 1875-1877.

Miotics have many disadvantages such as ciliary spasm, headaches, browaches, producing occasional precipitous rises in pressure, the development of tolerance by the treated eye, development of iris cysts, inducing retinal detachments, hypersensitivity to irritation reactions. In addition they require frequent instillations. These difficulties have stimulated many individuals in the past three quarters of a century to seek a better miotic or/and medical agent—one which produces none of these undesirable side effects and requires less frequent instillations. A variety of drugs which act directly on ocular structures, of autonomic agents, hormonal, osmotherapeutic, and so forth, have therefore been tried.

The exact site at which miotics produce the lowering of resistance to outflow has not been proved. It is thought that, in the narrow-angle glaucoma, the miotics are working mechanically to open the angle but, in chronic glaucoma, the real site of action has yet to be determined.

Adrenergic agents and adrenergic blocking agents are thought to produce their ocular effects chiefly by reducing inflow of intraocular fluid.

Anticholinesterase agents vary in their potency and length of activity as well as their side effects. A few new anticholinesterase agents have been given a trial in ophthalmology; some show potential value in the therapy of glaucoma. These include the phospholine iodide which has been tested at the Wills Eye Hospital and a new combination of two molecules of Neostigmine separated by the decamethyl group which has been reported on favorably by Gittler and Tillat.

Tranquilizing agents, antihypertensive and some of the diuretic agents will produce a temporary lowering of intraocular pressure. Experimental studies employing tonography have demonstrated the different sites of action of each of these compounds as recently studied.

A number of new carbonic anhydrase inhibitors are under study. The exact site and mechanism of these agents on aqueous humor dynamics has not been established, although several theories have been proposed and considerable data accumulated.

It is quite apparent that, within the last decade, many new agents for the control of intraocular pressure have been introduced.

One may now approach the problem locally and/or sytemically. Much more attention is being given to combine medication, not only of local agents but of local and systemically acting drugs. Local therapy is still of greatest importance. More attention has been devoted to therapy with systemic agents within the last few years. A great interest in the variety of agents which may influence intraocular pressure will probably lead to more information concerning the formation of aqueous humor and the dysfunctions which contribute to the development of a variety of glaucomas.

The problem of glaucoma in myopia

Dr. Hans Goldmann, Berne, Switzerland, said that older people with high myopia often have glaucoma without any of its symp-

toms. This form often is unrecognized, though it is not rare. To aid in the diagnosis of glaucoma in this type of patient, Dr. Goldmann has devised the applanation tonometer, which permits more precise measurements of intraocular pressure than the standard tonometer. Recognizing cases of glaucoma in such patients is of outstanding importance.

Jesse M. Levitt,
Recording Secretary.

MADRID OPHTHALMOLOGICAL SOCIETY

March 20, 1957

Film on cataract sutures

Dr. Aguilar Muñoz presented a film on "Continuous suture in cataract surgery." There was no discussion.

Diabetic retinopathy

Dr. Muñoz Pato described a case of diabetic retinopathy in a woman, aged 69 years. Four years previously, because of a persistent sciatic attack of two months' duration repeated urine and blood tests were done, with normal glycemia. During four years the sciatic attacks reappeared annually and further examinations remained normal.

One month ago the patient had a sudden loss of vision without pain, O.D. At examination, O.D. was in mydriasis without light reflex; vision was hand movements; tension, 16 mm. Hg. Ophthalmoscopy showed O.D., a large, bright red, circular hemorrhage occupying the macular region. The retinal vessels bent slightly over it and sloped down in passing it. On its superior border, the hemorrhage divided into smaller ones and numerous deep white foci were seen. The hemorrhage was located at a depth. There were discrete signs of sclerosis. Vision, O.S., was 1/3 and similar white foci were seen in the fundus. No hemorrhages were present.

The arterial pressure was 190/110 mm. Hg. Urinalysis showed slight albumin and glucose. Blood urea, 32; glycemia, 214; cholesterol, 196 percent.

Five days later the urine showed slight albuminuria; blood glucose, 142. The rest of the examinations were normal.

The case is interesting in that it presents an asymptomatic diabetes up to the moment of the retinal hemorrhage. It demonstrates how advanced the fundus changes can be without causing subjective troubles, until the macula is involved. Vitamins CP and B_{12} were prescribed. Glycemia decreased as soon as the patient kept a strict diet.

Discussion. Dr. Aguilar Muñoz: Because of the possible relation of the case presented by Dr. Muñoz Pato to a Junius-Kuhnt disciform macular degeneration, and to other macular degenerations, we suggest treatment with heparin.

It is recognized that arteriolar atheromas are produced by deposits of large lipoprotein molecules (B-lipoproteins) on the intima of the vessels. When the body has lost the ability to degrade the B-lipoproteins into small lipoproteins that can be well assimilated the atheroma occurs.

It may well be that macular degenerations are due to such deposits in the intima of the choroidal vessels. Heparin has the property of degrading the B-lipoproteins into A-lipoproteins and may help the macular degeneration cases.

In the case of Dr. Muñoz Pato heparin would be recommended, since the hemorrhage was not due to a blood dyscrasia but to alterations in the walls of the vessels (diabetes).

Dr. Muñoz Pato: I have thought of using heparin in this case as soon as the hemorrhage absorbed. Since the dosage required is below the anticoagulant level it may prove helpful.

Craniofacial dysostosis

Dr. del Rio Cabañas presented a boy, aged 14 years, who measured 1.48 meters in height and had a frontal sinking with

marked left facial hemiatrophy, causing a vertical asymmetry of the orbits and hypoplasia of the maxilla. In addition, he showed mental retardment, hypogenitalism, and discrete insipid diabetes. Exophthalmometer readings were: O.D., 25 and O.S., 29 mm. Hypertelorism of 40 mm. with convergent strabismus, due to paresis of both lateral recti, was observed. There was convergence dissociation. Vision was O.D., 1/3; O.S., 1/3.

Ophthalmoscopy revealed a marked pallor of both discs, diffuse choroidal atrophy with marked peripapillary atrophy of the choroidal vessels. Fields were reduced concentrically to 20 degrees, O.U. X-ray studies showed a marked thinning of the vault of the skull and digitations due to past endocranial hypertension. The Bertolotti syndrome was present —marked basilar lordosis with a ballooning of the sella. On profile, the skull showed a reduction of the anterior fossa with an enlargement of the posterior fossa, as would appear in premature union of the coronary suture.

Behçet's syndrome

Dr. Rio Cabañas and Dr. Calvo Pico presented a woman, aged 32 years, who in May, 1947, noticed marks on her ankles resembling mosquito bites which disappeared in three days and reappeared three months later. During this time she suffered buccal and genital aphthae and was treated with anatoxine, calcium, and sincrobin. Her condition improved but her eyes became irritated, painful, and the lids were swollen. Her vision decreased.

In December, 1956, her right eye had to be enucleated because of glaucoma secondary to a pathologic cataract.

At examination, O.S. had vision reduced to counting fingers at one meter. There was no pupillary reflex and the pupil was fixed from synechias. In January, 1957, iridocyclitis with hypopyon appeared.

The blood showed slight hypochromic anemia, leukocyte reading of 5,300; vs: 22 mm. in the first hours' urine, normal.

Treatment with achromycin (250 mg., every six hours) Gevral, sincrobin and vitamin B-complex helped the patient, and 15 days later vision, O.S., was 1/3. Her weight increased. In March there was a relapse with aphthae, tonsilitis, and hypopyon. Vision dropped to finger counting at one meter. Again with the same treatment there was some improvement.

Discussion. Dr. Bertolotti referred to a case of Santa Cruz and pointed to the possibility that all these oculomucocutaneous syndromes were caused by a neurotropic virus.

Dr. del Río Cabañas mentioned that Sezer, when he discovered the virus of Behçet's syndrome, reported on the neurotropic properties of the virus which causes an encephalomeningomyelitis in the experimental animal. According to him the ocular manifestations would start in the optic nerve and retina and later involve the uvea, giving rise to the relapsing attacks of uveitis with hypopyon.

Conjunctival capilloroscopic index

Dr. Río Cabañas and Dr. Calvo Pico studied the conjunctival capilloroscopic index in 150 patients which were classified into five groups as follows:

1. Glaucomatous patients (40).
2. Arteriosclerotic patients (20).
3. Hypertensive patients (50).
4. Nephritic patients (30).
5. Normal patients (10).

In the glaucomatous patients, the average index was 36.2; with tensions above 39 mm. Hg, 39; above 25 mm. Hg, 36.5; below 25 mm. Hg, 30.

In the anteriosclerotic patients, the average index was 45.5; in the hypertensive patients, 51.3; with arteriol pressure above 20, 56.5; with arterial pressure below 20, 47.5.

In the nephritics the average index was 10.1. The normal group had an average index of 18.2.

Olga Ferrer,
Translator.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

Address original papers, other scientific communications including correspondence, also books for review to *Dr. Derrick Vail, 700 North Michigan Avenue, Chicago 11, Illinois;* Society Proceedings to *Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Manuscripts should be *original copies,* typed in *double space,* with wide margins.

Exchange copies of the medical journals should be sent to *Dr. F. Herbert Haessler, 561 North 15th Street, Milwaukee 3, Wisconsin.*

Subscriptions, application for single copies, notices of changes of address, and communications with reference to advertising should be addressed to the *Manager of Subscriptions and Advertising, 664 North Michigan Avenue, Chicago 11, Illinois.* Copy of advertisements must be sent to the manager by the 10th of the month preceding its appearance.

Change of address notice should be received not later than the 10th of the month prior to the issue for which the change is to go into effect. Both old and new addresses should be given.

Author's proofs should be corrected and returned within forty-eight hours to the *Manuscript Editor, Mrs. Katherine F. Chalkley, Lake Geneva, Wisconsin.* Fifty reprints of each article will be supplied to the author without charge. Additional reprints may be obtained from the printer, the George Banta Company, Inc., 450-458 Ahnaip Street, Menasha, Wisconsin, if ordered at the time proofs are returned. But reprints to contain colored plates must be ordered when the article is accepted.

THE NEW CALIBRATION SCALE FOR SCHIØTZ TONOMETERS

The Committee on Standardization of Tonometers of the American Academy of Ophthalmology and Otolaryngology at its annual meeting on October 12, 1956, adopted a new calibration scale for Schiøtz tonometers and named it the scale of 1955. The scale had been submitted by the late Dr. Jonas S. Friedenwald with the request for further rigorous empirical testing with several standardized tonometers. These tests showed the 1955 scale to be highly self-consistent, that is, conducive to concordant readings with two or more different weights. Very much encouraged by this finding of consistent self-consistency, the committee agreed with Dr. Friedenwald that "the new

scale probably represents a closer approach to the truth" and published it in the January-February issue of the *Transactions* of the American Academy of Ophthalmology and Otolaryngology.

The reactions to the new scale which have reached my desk have, on the whole, been indicative of approval and relief, but there have been expressions of dismay, alarm, and suspicion, such as: "Why still another scale?" . . . "Isn't there enough confusion already?" . . . "Why not let us all go back to the 1948 scale which served as well?"

There are answers to these questions. The 1955 scale was a necessity in the sense that it corrected a serious flaw in the 1954 scale. Unfortunately, this flaw was not recognized before the official adoption of the scale. The committee and I, in particular, take the blame for this oversight. Recognizing the 1955 scale as a necessary sequel to the 1954 scale, the ophthalmologist has the right to ask: What were the reasons for adopting the 1954 scale and what did the Committee on Standardization of Tonometers hope to accomplish by changing from the 1948 scale to the 1954 scale?

The answer is that the committee believed the 1954 scale to be a better estimate of the true intraocular pressure than any scale advocated previously. The advent of tonography, and particularly the fact that we had learned to measure the pressure in the episcleral veins accurately, created a new demand for the best possible estimate for the intraocular pressure. Needless to say, the 1954 scale was almost entirely based on the calibration studies of the late Dr. Jonas S. Friedenwald, who summarized his work in the Decennial Report of the Committee on Standardization of Tonometers, published in 1954. While this report brought out, with almost painful clarity, the many difficulties and uncertainties in the procedure of calibration, it also showed Friedenwald's ingenuity and resourcefulness at work, devising new approaches to the various aspects of the problem.

The principal task in tonometer calibration is to determine the pressure in the undisturbed eye (P_o) which corresponds to the new pressure brought on by the application of the tonometer and recorded by it (P_t, the pressure during tonometry). In the actual process of calibration an enucleated eye, suitably suspended or bedded down, is connected with a saline reservoir and a manometer by means of a cannula introduced into the anterior or vitreous chamber. At various pressure levels produced by adding saline to the contents of the eye, simultaneous tonometric and manometric readings are taken. If the connection between the eye and the manometer is kept open during the tonometry, "open stopcock" or P_t values are recorded. If the stopcock is closed before the tonometer is applied, the manometer records P_o or a pressure that very closely approaches P_o. The crux of tonometer calibration lies in that during these P_o measurements the ocular rigidity factor (K, coefficient of ocular rigidity) manifests itself in a most disturbing magnitude. At the same manometric pressure the tonometric reading varies grossly from one eye to the next and, in the same enucleated eye, from one hour to the next. These variations can only be attributed to changes in the distensibility or its reciprocal, the rigidity, of the ocular coats. The magnitude of these changes seems to be greater post mortem and under the stresses entailed in the calibration procedure than in vivo.

A solution which might occur to the reader at this point would be to estimate the ocular rigidity prevailing at the time of the P_o measurement by observing the rise in pressure resultant from small, accurately measured increases in the volume of the ocular fluid contents. A correction factor might then be applied to the P_o values to make up for the difference between post mortem and in vivo ocular rigidity. Dr. Friedenwald considered this possibility but gave it up because of the extreme variability of the P_o values in his and his technicians' hands, and

also for reasons that had to do with his instrumental setup.

In sharp contrast to these P_o determinations, the P_t measurements made in his laboratory on animal and human eyes were highly consistent and reproducible and, therefore, became the main pillar of Dr. Friedenwald's calibration method. The idea underlying his method was this: The rise in intraocular pressure from P_o to P_t is due to the displacement of intraocular fluid, that is due to the indentation of the cornea by the plunger of the tonometer. If the volume of this indentation (V_c) could be measured and if the general relation between intraocular fluid volume and pressure were known, it should be possible to calculate P_o from the observed P_t values. Friedenwald had derived a formula for the volume-pressure relationship from the work of earlier investigators. For the measurement of the volume of the corneal indentation (V_c) he used a method consisting of clamping the anterior segment of human and animal eyes onto fluid-filled chambers with an air-bubble trapped in the fluid column leading to the manometer. The air-bubble indicated the amount of fluid displacement that occurred upon application of the tonometer.

To make his calculated P_o values applicable to living eyes, Friedenwald needed an estimate of the average ocular rigidity as it prevails in living eyes. This he obtained from a large series of paired tonometric readings that had been made in his office a good many years before with a tonometer which was then considered a standard instrument. The mean scale reading was 4.03 with the 5.5-gm. weight and 9.89 with the 10-gm. weight.

The P_o values calculated from these P_t, V_c, and K measurements are shown in the table as the scale of 1954, together with Schiøtz's scale of 1924 and the committee's scale of 1948. For the 5.5- and the 7.5-gm. weight the new scale gives, on the whole, lower P_o values than the older scales.

During the early part of 1954 the scale of that year received confirmation from two independent sources, at least as far as the 5.5-gm. weight was concerned. With a fine instrumental setup Dr. Morton S. Grant had made some simultaneous P_o and ocular rigidity (K) measurements on enucleated human eyes. Correction of these P_o values for average, in vivo, ocular rigidity yielded P_o values in very close agreement with Friedenwald's 1954 scale. Another confirmation came from Goldmann's clinic. With his applanation tonometer which gives estimates for the intraocular pressure almost entirely free of the rigidity factor, the average pressure of the normal human eye was found to be 15.45 ± 2.5 mm. Hg. The average normal tonometric scale reading with Schiøtz tonometers and the 5.5-gm. weight is 5.25 scale units which, according to the 1954 scale, is equivalent to 16.7 mm. Hg, only 1.25 higher than Goldmann's figure.

With these confirmations the P_o values of the 1954 scale for the 5.5-gm. weight seemed to be well founded when the scale was adopted by the committee. Since then no experimental or clinical findings have been disclosed which have shaken the committee's belief in the scale for the 5.5-gm. weight. Even the recent calibration work by Earle McBain may be interpreted as a confirmation of the 1954 scale insofar as the values for the 5.5-gm. weight are concerned. Combined applanation and indentation tonometry in Goldmann's hands has confirmed Friedenwald's estimate of the average ocular rigidity. All this concordant evidence may serve as an answer to the question asked at the outset: Why did the Committee on Standardization of Tonometers supplant the 1948 scale with the 1954 scale?

It did not take long to discover the serious flaw in the 1954 scale, that is, the unfortunate discrepancy within itself which causes the pressure estimate to be higher with the higher plunger loads. Nor did it take long to find flaws in some of the measurements that were the basis of the 1954 scale. The results of Friedenwald's paired readings on living eyes could not be duplicated. Unfortunately

CONVERSION TABLES FOR SCHIØTZ TONOMETERS

Scale Reading	Loads in gm.											
	5.5				7.5				10.0			
	1924	1948	1954	1955	1924	1948	1954	1955	1924	1948	1954	1955
0	48	44	40	41	67	62	58	59	91	83	82	82
1	41	37	34	35	57	53	50	50	77	72	71	69
2	35	32	28	29	49	46	43	42	66	63	61	59
3	30	27	24	24	42	40	37	36	57	55	53	51
4	26	23	20	21	36	35	32	30	49	48	47	43
5	22	20	17	17	31	30	27	26	42	43	41	37
6	19	17	15	15	27	27	24	22	36	37	35	32
7	17	14	12	12	23	23	20	19	31	33	31	27
8	15	12	10	10	20	20	17	16	27	29	27	23
9	13	10	9	9	18	17	15	13	23	26	23	20
10	12	8	7	7	15	14	13	11	20	22	20	17
11	10	6	6	6	14	12	11	9	18	19	17	14
12	9	4	5	5	12	10	9	8	15	17	15	12

there is no record of the physical characteristics of the Schiøtz tonometer with which Friedenwald took those readings. The instrument itself has long been lost. P_t measurements by McBain and Grant have yielded results quite different from those of Friedenwald's as far as higher weights are concerned. Fortunately for ophthalmology, Dr. Friedenwald was made aware of some of these discrepancies before he became seriously ill. He was certainly best qualified to judge what interpretations other than those incorporated in the 1954 scale his experimental data permitted. Self-consistency of the scale, that is—to say it once more—concordant readings with different plunger loads, was most important to him, the ardent advocate of paired tonometric readings as a method of estimating ocular rigidity. Guided by a large series of such paired in vivo readings he repeated the process of fitting curves to his experimental data and arrived at new constants which yielded the self-consistent P_o values that make up the 1955 scale. It differs from the 1954 scale in that the pressure estimates for the higher weights have been reduced.

If this all sounds like a thorny and devious path to a precarious ledge just half way up the mountain, it may help to remember the principal points:

1. The clinically most important part of the new scale, that is, the part concerning the 5.5-gm. weight, is supported by three independent and altogether different methods, applanation tonometry (Goldmann), direct calibration (Grant), and indirect calibration (Friedenwald).

2. The principal aim in working out the scales for higher weights was self-consistency. This has been achieved in the 1955 scale to an unprecedented degree and for the tonometers now in general use.

3. The meaning of the tonometric reading per se has not been changed in any way. With a standardized Schiøtz tonometer and the 5.5-gm. weight a reading from 5.0 to 5.5 scale units still represents the average for normal eyes, and a reading of three scale units still represents a borderline value, very rarely encountered in normal eyes, assuming, of course, average ocular rigidity.

4. The large series of paired readings used in the preparation of the 1955 scale and summarized by me in the January-February issue of the *Transactions* of the American Academy of Ophthalmology and Otolaryngology will serve as a firm basis in the valuation of any set of paired readings. This new yardstick should give the ophthalmologist new confidence in paired readings as a means of estimating ocular rigidity.

Thorny and devious as the path of tonometer calibration has undoubtedly been, the

present position seems reasonably secure and not too far from the summit.

Peter C. Kronfeld.

CORRESPONDENCE

Iridectomy for essential atrophy of the iris

Editor,
American Journal of Ophthalmology:

In a recent article, "Iridectomy for early essential atrophy of the iris," by Louis Daily and Ray K. Daily (The Journal, 44:487-492 [Oct.] 1957), the authors state: "We know of only one case other than that reported here in which iridectomy was performed prior to onset of glaucoma." Due credit for the pioneer case is given Derrick Vail who remarked in a few lines of editorial comment in *The Year Book of Eye, Ear, Nose, and Throat*, 1955, that iridectomy controlled the tension in his patient for an observed period of eight years. I should like to record a third case:

A 30-year-old woman visited me in March, 1957, with the complaint that the pupil of the left eye had been wandering upward for nine years and that the eye had been recurrently irritable. She was now having difficulty in doing her work, modelling clothes for photography, because of extreme photophobia. Examination disclosed that the left pupil was contracted, vertically pear-shaped, and drawn up to the corneal limbus; and that holes had formed in the iris temporally and inferonasally, simulating additional pupils. The iris showed a loss of topography. The pupil reacted to light and the anterior chamber was of normal depth. The left cornea had diffuse punctate opacities from recurrent attacks of bullous keratitis. Fluorescein revealed an irregularly distributed punctate staining.

Her unaided acuity was: R.E. 20/13; L.E., 20/70 (with pinhole, 20/25). The ocular tension was: R.E., 16 mm. Hg; L.E., 16 mm. Hg. After instilling a mydriatic in the left eye, the pupil became rounded but was still somewhat drawn up, and the ocular tension remained unchanged. Gonioscopy showed the temporal side of the angle free, but peripheral synechias elsewhere.

In May, 1954, she had consulted Dr. H. Wyatt Laws of Montreal who had diagnosed essential atrophy of the left iris and left superficial punctate keratitis. He found tonometry, tonography, and gonioscopy negative for glaucoma.

I wrote Dr. Parker Heath about the case and received a prompt and detailed reply, in which he said: "As for the best treatment, in anticipation of ultimate glaucoma, I would favor iridectomy. It seems to me that your case is not one of the cuticular membrane variety. This means that the glaucoma when it comes will be due to anterior synechiae blockade of the angle plus the factor of less iris area for absorption. The obstructive factor is the more important. The point of attack should be at the iris base toward which the pupil is migrating. The iris always bunches up on the strong side of the dilator myoid and from here the anterior peripheral synechias fan out around the arc of the angle. The dilation of the pupil seems to have a good effect upon your case which suggests that in this instance the uveitis factor may be of considerable importance."

The bullous keratitis was temporarily controlled by instillation of Antrenyl (one percent) (an atropine substitute), sodium chloride (two percent), and Chloresium; then more effective relief was obtained with the additional instillation of Dionin (five percent) in the morning.

The necessity for surgery was discussed and consultation with Dr. Derrick Vail was arranged. He advised cyclodialysis with iridectomy in the upper nasal sector. The operation, which followed the Wheeler technique, was done on July 5, 1957, and the patient was discharged from the hospital after four days.

Since the operation the pupil has dilated more fully and the corneal edema and photophobia have completely subsided. At the last visit, October 26, 1957, the ocular tension was: R.E., 17 mm. Hg; L.E., 17 mm. Hg. Scattered minute macular opacities were still present on the cornea and had shown no attenuation after a two-month trial of hydrocortisone ointment and Diamox (250 mg. once daily). The correction of the left eye under cycloplegia was: +1.0D. sph. $\bigcirc$ +1.0D. cyl. ax. 100°, 20/25— (with pinhole, 20/20—). The patient was highly gratified with the operative result.

In this woman the iris of each eye was a definite brown. The authors comment that in 10 cases in which the iris color was noted, the irises were gray-blue, gray-green, or gray, and suggest that the disease may have a predilection for gray irises. My case would indicate that the iris color is not significant.

(Signed) James E. Lebensohn,
Chicago, Illinois.

BOOK REVIEWS

CLINICAL NEURO-OPHTHALMOLOGY. By Frank B. Walsh, M.D. Baltimore, The Williams & Wilkins Company, 1957, second edition. 1,294 pages (triple column), 441 illustrations, references, index. Price: $29.00.

Dr. Walsh, associate professor of ophthalmology, The Johns Hopkins University, has given us a second and more elaborate edition of his classic book, the bible of neurologists, neuro-ophthalmologists, and even plain ophthalmologists, first published 10 years ago. A successful attempt was made by the author to add much new and important information and to eliminate the few errors of the first edition.

The format and arrangement of the first edition are followed, but the size and weight are increased, a doubtful advantage because edition one weighed seven pounds and edition two weighs eight and one-half pounds (something like holding a newborn baby on your lap when you read it). The double column of the first edition is now increased to three columns to a page and I found it somewhat more difficult to read on this account. However, the scientific contents more than make up for this trivial criticism.

The meticulous life work in this field by Dr. Walsh is reflected on every page. His clinical investigations and voluminous reading assure us of his great authority and we cannot do without his book for reference and study in our daily tasks. This book is truly a classic and we are most proud that the author is one of us.

Derrick Vail.

REHABILITATION IN ENGLAND. With a Report on the European Seminar for Rehabilitation of the Blind. Sponsored by the Ministry of Labor, Bonn, 1957. 287 pages, 41 illustrations (Arbeit und Gesundheit fasc. 62) Price: DM36.-.

Under the sponsorship of the Ministry of Labor of Western Germany, a number of experts on rehabilitation studied some of the institutions and their methods of treatment in England, with the view of gaining additional information for planned revisions in the German legislation.

Prof. W. Rüsken devotes a chapter to the social integration of disabled persons in England. His report is based on his observation of a number of institutes for general and specialized rehabilitation. Even in hospitals, emphasis is placed on rehabilitation until such time when the condition of the patient permits transfer to one of the regular rehabilitation centers. It has been the experience that institutional treatment is preferable to ambulatory therapy, although the latter has a place in big cities. Since the second World War 420 sports divisions, 300 physiotherapeutic, and 200 work-therapeutic divisions have been added to the existing hospital facilities. There is a tendency to avoid grouping patients suffering from the same disease or injury together because discussion of their ailments may interfere with their progress. The average stay in the center is three months.

Patients who are not expected to be rehabilitated in such a relatively short period of time or who cannot resume active occupations are not brought to industrial rehabilitation units but rather to government training centers. Such patients are double-amputees, hemiplegics, patients with severe tuberculosis, and so forth.

P. Kerschbaum summarizes the English rehabilitation system. His review concerns itself with the legal principles; the constitution of various types of units in the rehabilitation service; a detailed description of representative centers in the various categories; and the organizational, social, and psychologic problems of rehabilitation.

The basic law is the Disabled Persons (Employment) Act, 1944, an outgrowth of the Tomlinson report of 1941. Handicapped, in the legal sense of the word, is anybody who is limited because of trauma, disease,

or a congenital malformation. Such a person will get supplementary aid to make up the difference between his actual and theoretic earning capacity.

The purpose of government training centers is the rehabilitation of disabled persons who need schooling for a different occupation. The average stay there is six months. At present there are 22 government training centers in operation; whereas, the large number of war injuries made it necessary to run 78 such centers in 1947. In addition to these institutes established by the government, there are semigovernmental, industrial, and charitable institutions. Although there is still a considerable reluctance to absorb handicapped or disabled persons in general, some factories will accept as much as a five to 10 percent contingency.

Garston Manor Rehabilitation Center is described in some detail as a specific example of a government center created on the initiative of the Ministry of Health.

Roffey Park Rehabilitation Center for psychoneurotics was opened under the tutelage of the National Council for Rehabilitation of Industrial Workers after the large manor had been acquired with contributions from 175 firms.

Rehabilitation centers for tuberculous patients are examples of industrial centers. Some are ambulatory institutes—mostly in big cities, like the Royal Chest Clinic in Birmingham, with patients living at home—or they are sanitaria. An example of the latter is Papworth.

Very interesting work for the rehabilitation of spinal injury cases is done in the Stoke-Mandeville Hospital in Aylesbury.

Kerschbaum describes a number of other centers in considerable detail.

Drs. Kreusch, Lemberg, and Volkmann base their exceedingly interesting report about medical and surgical methods on their studies conducted exclusively in the Rehabilitation Center for Spinal Injuries in Stoke-Mandeville, during the period June 1 and August 31, 1956.

The last chapter of the book is of particular interest to ophthalmologists. It covers the proceedings of the European Seminar for the Rehabilitation of the Blind which was arranged by the World Council for the Welfare of the Blind together with the World organization of front-line soldiers and the co-operation of the United Nations and the International Labor Organization. In addition to representatives from 18 European nations, there was a speaker from the United States and one from Canada. The seminar concerned itself not only with blind veterans but also with civilian casualties.

There seems to be a growing tendency in almost every country to rehabilitate the blind rather than just to give them a dole. There are understandable differences in the approach, and some countries have gone further than others. But almost everywhere it seems to be a thing of the past to consider some menial tasks like basket weaving, making brushes, and so forth, as the equivalent of a gainful occupation for the blind.

The Rt. Hon. Iain Macleod, speaking for Great Britain, gave a brief historical review leading up to the present situation. He touched on the legislation regarding the blind. College students are aided to complete their studies. College graduates are enabled to start or continue their profession.

G. C. Slater from the British Ministry for Labor talked about co-operation between private and official organizations for the welfare of the blind. A Blind Persons Act already existed in 1920. Since the enactment of the Disabled Persons Employment Act of 1944, the role of the state has become all important. The local school authorities are responsible for the professional training—in England to the age of 21 years, in Scotland to the age of 18 years. The Ministry of Education makes some financial contribution. The Ministry of Labor is responsible for the rehabilitation of adults. In addition to these official organizations, some private groups carry out rehabilitation. Qualified persons may get grants for a college education.

J. C. Colligan, general secretary of the

Royal National Institute for the Blind, chose the same topic as the preceding speaker. The main task of the institute he represented is to provide the 100,000 blind in Great Britain with books in braille, with apparatus and aids for the blind, and with certain schools. The institute is interested in the foundation of recreation centers and vacation homes, in rehabilitation centers for the recently blinded, and in employment facilities.

Sir Ian Fraser, president of the St. Dunstan's Organization for Men and Women on War Service, stressed that newly admitted persons find it much easier to adapt themselves to their new situation by living together with blind teachers and others who have already conditioned themselves to their blindness. The main task of his organization is the psychologic adjustment of the blind.

L. Fawcett, of the same organization, spoke on the facilities and the physical makeup of St. Dunstan's.

Russell C. Williams, director of the Rehabilitation Department of Hines Veterans Hospital, Hines, Illinois, discussed the methods of his center. There is no abstract of his talk.

Gérard Borré, expert for the United Nations and president of the Braille League in Brussels, talked on specialized industrial concerns that protect the blind against competition of normal workers. It should be the responsibility of the state or charitable institutions to enable the blind to work in such surroundings—either permanently if they are unable to meet free competition or at least until a time when they are prepared for such competition. Although it is quite often difficult to put such industrial enterprises on a self-sustaining basis, they are a preferable solution to the mere training of the blind to do handicraft at home or to support them outright.

Ernst Jorgensen spoke about the occupation of the blind in industry and commerce in Denmark; L. F. Chester chose the same topic regarding Great Britain.

Col. E. A. Baker, chief executive for the Canadian National Institute for the Blind, discussed in a very informative manner the Canadian approach to the problem. A field secretary is responsible for a certain geographic area of from 350 to 600 registered blind. His task is to register all new cases of blindness and to initiate procedures to get them to the right place. Field secretaries have special training for this work, just like the employment agents. The latter not only have to find suitable places of employment for the blind but also to check these places regularly and eliminate any possible difficulties. A most enlightening part of the speaker's talk was devoted to the curriculum for teachers for the blind.

This is of necessity not a complete list of all the talks given at the seminar but it should suffice to form a general impression of the tenor of this most important convention.

There is a very appropriate listing of the facilities for the rehabilitation of the blind for the following countries: Austria, Denmark, Finland, France, Germany, Great Britain, Greece, Eire, Italy, Holland, Norway, Portugal, Spain, Sweden, Switzerland, Turkey, and Yugoslavia.

Stefan Van Wien.

THE GLAUCOMAS. By H. Saul Sugar, M.D. New York, Hoeber-Harper, 1957. 497 pages, 164 figures, extensive bibliography and index. Price: $13.50.

The author, who is the director of the Glaucoma Clinic, Receiving Hospital, Detroit, and assistant professor of ophthalmology, Wayne University Medical School, published the first edition of his work in 1951. It was widely and well received. At first glance, it would not appear that there had been enough advances in our knowledge of glaucoma in the past six years to warrant a new edition. It is most gratifying to realize that this impression is false, for these past years have witnessed further important developments in tonography, use of Diamox, surgical procedures, and even new anatomic conceptions among others. Everett Kinsey

assisted the author in the rewriting of the chapter on the aqueous and Bernard Becker contributed to the chapter on tonography. The chapter on drugs was revised with the advice and collaboration of Mr. Robert Feinstein. The various surgical procedures and techniques are well described and illustrated and more generously than in the first edition.

This book is a sound and most valuable contribution to a difficult subject. It is fairly and lucidly written, authoritative and modern. The printing and illustrations are excellent. It is highly recommended.

Derrick Vail.

THE VERTEBRATE VISUAL SYSTEM. By Stephen Polyak, M.D. Chicago, the University of Chicago Press, 1957. 1,390 pages, 546 illustrations, index, bibliography. Price: $45.00.

This book is the result of 30 years of "almost continous labor" and "originated in the desire to understand better the anatomical structures underlying vision, the way in which they work, and how they originated." As pointed out, it was the intent of the author to write not only for the scholar and advanced investigator but also for the student and beginner in neuro-anatomy, neurophysiology, clinical neurology, ophthalmology, and related fields.

During the last few years of Dr. Polyak's life he was in ill health and was working against sudden death and blindness. He died while the book was in the process of publication and his long-time friend, Heinrich Klüver, spent almost two years of constant work to complete the task.

The book is divided into four major parts:

Part 1, composed of four chapters, is on the history of investigations of the structure and function of the eye and of the visual pathways and centers of the brain. It covers the work done from the time of Aristotle and Hippocrates to the modern investigators. The chapters are on the history of the investigations of the structure and function of the eye and the development of the optical sciences; investigation of the visual pathways and centers during classical antiquity, the middle ages, and the early period of the modern scientific era; investigation of the visual pathways and centers during the early period of modern brain research; and modern anatomic experimental, clinical, and pathologic investigation of the visual pathways and centers.

The second part, consisting of six chapters, deals with the anatomy and histology of the retina and the visual pathways and centers of the brain; optic nerves, chiasma, tracts, and subcortical centers; visual radiation; cortical visual centers; general theory of organization of the nervous system and the visual function; and the blood supply of the vertebrate visual system.

Most of the material on the vertebrate retina had been presented previously in a book, *The Retina*, which was published in 1941.

The third part of the book, composed of two chapters, deals with the pathology of the retina and the visual pathways and centers of the brain. Of the two chapters, one covers the disturbances of vision and the anatomic organization of the visual system while the other discusses the clinical and pathoanatomic observations of cases with disturbances of visual fields.

In the fourth part of the book, consisting of three chapters, is an account of the origin, development, comparative anatomy, physiology, and biology of the vertebrate organs of sight. In this section the origin and development of the vertebrate eye and visual system, and vision and its role in the origin of man are discussed.

There is an epilogue which consists of a retrospect on the role of vision and other factors in the origin of man together with a speculation on the future of mankind.

In the last few paragraphs of the book one senses the value of vision as felt by the almost blind author when he states: "Vision just like life is a heavenly gift bestowed upon

man for whatever use he is capable of putting it to. With it we are able to see and appreciate and understand countless objects necessary in our daily life and make use of them in practical ways. . . . Vision makes us see and appreciate the smile on a friendly face, enjoy the endless beauties of nature, the multicolored flowers, birds, and fruits, the green meadows and somber deserts, the glorious sunrise and sunset, the majesty of the tall mountains and the sweeping horizon of the ocean, the magnificence of an Alhambra and of a Taj Mahal, and other things which make our visible world."

Appended is a bibliography of 299 pages which contains references on the vertebrate visual system from the time of Aristotle (4th Century B.C.) to the present. This is of inestimable value to the research worker or the serious student of this subject.

There is an excellent, comprehensive index of 31 pages, including reference not only to subject but also to authors.

To the research worker in the visual field using animals, the anatomic details and physiology of the visual apparatus with which he is working is of great importance if conclusions are to be drawn in regard to the human system. The information contained in this volume make it invaluable for such a worker.

The format is excellent. The paper is of good quality and the print easily readable. The illustrations are superb, both black and white and color, and represent the best in the engraver's and printer's art. In view of the fact that it is a large volume and that it will be used extensively as a reference one wonders whether or not the binding is strong enough to withstand such use.

The publication of this valuable book was made possible by grants from the Dr. Wallace C. and Clara A. Abbott Memorial Fund of the University of Chicago; the John Simon Guggenheim Memorial Foundation; the United States Public Health Service; and the National Science Foundation.

It is impossible to review such a monumental work adequately in the limited space available. Suffice it to say that the book is an essential for every laboratory dealing with the subject. In addition every scholar or serious student in the field of the visual system cannot afford to be without it. It is destined to become a classic in the field of the vertebrate visual system and should be in every medical library.

Frederick C. Cordes.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

7

CONJUNCTIVA, CORNEA, SCLERA

Sachs, A. **A new medium for the storage of donor eyes for corneal grafts.** Brit. J. Ophth. **41**:558-561, Sept., 1957.

The increasing demands for donor eyes and the disadvantages of present storage facilities for these eyes have resulted in the preparation of a new storage medium which possesses advantages over previous media in that the eyes remain or become more nearly sterile and are physiologically more suitable for transplantation. The enucleated eyes are immediately immersed in the solution which contains intradex, dextrose, phenol red, chloramphenicol, streptomycin and nutrient agar and may be stored in refrigeration for ten days before use. (1 reference)

Morris Kaplan.

Somerset, E. J. **Mooren's ulcer treated by diathermy coagulation.** Brit. J. Ophth. 41:570-573, Sept., 1957.

Corneal irritation for three months in a 25-year-old man was the result of a typical advancing Mooren's ulcer which was first treated with atropine and patch. After no improvement, a delimiting keratotomy was done and this was followed by several cauterizations with carbolic acid and later with beta irradiation, all without improvement. The ulcer was then cauterized by diathermy coagulation which caused considerable improvement though two small areas persisted. These had to be cauterized a total of five times but eventually healed. (9 figures, 7 references)

Morris Kaplan.

Tosch, Cäcilie. **The treatment of epidemic keratoconjunctivitis with a hydrocortisone-neomycine ointment.** Klin. Monatsbl. f. Augenh. **131**:522-526, 1957.

If the treatment is initiated before the fourth day of the disease, a corneal lesion is rarely observed. Without this treatment 70 percent of the affected patients develop corneal lesions. 31 references)

Frederick C. Blodi.

Voinova, T. **Combating of trachoma in the U.S.S.R. in 40 years.** Vestnik oftal. **5**:20-29, Sept.-Oct., 1957.

The control of trachoma in U.S.S.R. in the period before the revolution was a charitable project and was not maintained by the state. As early as 1926 the Soviet Health Ministry provided am-

bulatory service for trachoma patients. A number of trachoma institutes were established for the study of the disease. This study led to the opinion that the etiologic factor of trachoma was a virus; an allergic component, a chronic inflammatory process of a proliferative character, may also play a part.

As a result of intensive treatment the number of trachomatous patients is only 6.7 percent of all the eye patients. The majority of the patients have trachoma stage III. Treatment takes only from two to three months now rather than three or more years. The disease is less severe; the cornea is not affected severely, the scars in the conjunctiva are not marked. The control of the recurrence is still important in regions where the disease is endemic and the treatment is continued a few months after the clinical cure.

Olga Sitchevska.

8

UVEA, SYMPATHETIC DISEASE. AQUEOUS

Algan, B., and LeGrand, P. **Rheumatic uveitis following cataract operations.** Bull. Soc. belge d'opht. **115**:257-260, 1957.

Insidious, intraocular inflammations still follow uncomplicated cataract extractions occasionally. A specific type of post-surgical unilateral uveitis in patients with chronic rheumatoid arthritis is described. It starts 3 to 20 days after an uneventful operation. The characteristic signs and symptoms are loss of the anterior chamber, extreme hypotony and a torpid course. In spite of the absence of pain or injection of the eyeball the presence of plastic exudate may lead to seclusion and occlusion of the pupils. Early preventive treatment, preferably three days before the operation and extending up to eight days after the operation should be given routinely to carriers of rheumatoid disease. Among 300 cataract extractions this type of uveitis was seen five times (1.7 percent) and the individual complications and their treatment are described. (10 references)

Alice R. Deutsch.

Anderson, B. and O'Neill, J. **Malignant melanoma of the uvea.** A.M.A. Arch. Ophth. **58**:337-347, Sept., 1957.

Fourteen cases of stationary or slow-growing malignant uveal melanoma are presented. The author discusses the effect of environmental factors and suggests that future control of these tumors may be related to the recognition and control of these factors. If we could measure the growth potential we could the better decide about the urgency of enucleation. (11 figures and 2 references)

G. S. Tyner.

Appelmans, M., Michiels, J. and Alearts, R. M. **Anterior chamber lipoidosis in iritis diabetica.** Bull. Soc. belge d'opht. **115**:219-228, 1957.

Lipoidosis of the anterior chamber is of rare occurrence. It is manifest, occasionally, as a sign of severe local tissue destruction in eyes with absolute glaucoma, in severe circulatory embarrassment following carotid sinus aneurysms and late intraocular tumors. It also may accompany general metabolic disturbances such as hypothyroidism, hypopituitarism and diabetes. The abnormal components of the aqueous in diabetic iridopathy and the clinical appearance of the aqueous in diabetes are discussed. The gelatinous plastic exudate, visible in the anterior chamber, disappears readily after insulin therapy. In contradistinction to the xanthomatous exudate in xanthomatosis bulbi which only disappears slowly, it is independant of retinal disease.

The anterior chamber content of a 50-year-old diabetic woman with severe hypercholesteremia, recurrent iritis and several attacks of diabetic coma showed the viscosity characteristic for diabetic iritis, elevated albumen content and an

increased amount of neutral fats and cholesterol. The albumen- cholesterol ratio equalled 4/1; 123 degenerated cells were seen in 1 mm. The transitory nature of these aqueous changes and the similarity of the clinical and chemical abnormalities of the aqueous in human diabetics and the aqueous in experimental alloxan-diabetic rabbits are briefly outlined. (1 table, 17 references)

Alice R. Deutsch.

Emmrich, K. **Suturing of the iris.** Klin. Monatsbl. f. Augenh. **131**:350-352, 1957.

A peripheral iridectomy is enlarged, if necessary, with an iridotomy down to the pupil. The cleft in the iris is then closed with one or two 5-0 Perlon sutures. (2 figures) Frederick C. Blodi.

Johnson, D. S. and Pino, R. H. **Keyhole and peripheral iridectomies in different eyes in the same patient.** A.M.A. Arch. Ophth. **58**:421-425, Sept., 1957.

Fourteen cases are reported. There was no difference in subjective glare in the two eyes in all but two cases. There was no difference in bulge of the hyaloid face and the visual results were slightly better with the iridectomy. The lids covered the iridectomies so well that cosmetically there was little difference. There was slightly greater astigmatism with the keyhole operations. (1 table, 7 references)

G. S. Tyner.

Quiroz Salgado, L. and Cavalcante Queiroz, A. **Essential progressive atrophy of the iris, a case report.** Arq. brasil. de oftal. **20**:41-52, 1957.

Essential progressive atrophy of the iris occurs as a unilateral disease, found most frequently between the ages of 20 to 45 years, and apparently affecting women in a ratio of 5 to 1. It has been observed in the white race exclusively. Initially pupillary changes occur: elongation, ectopia and small peripheral anterior synechiae. Zones of iris atrophy soon follow, located opposite the direction of pupillary deviation. This atrophy involves the ciliary portion of the iris, sparing the sphincter and pupillary zone. The stroma is affected first, then the posterior pigmented layer, with the clinical appearance of holes in the iris through which the equator of the lens, the zonule and the ciliary processes may be seen. Ectropion of the pupillary margin also occurs. The iris holes increase in size and number until the true pupil is barely discernible, while the peripheral anterior synechiae increase in size and density. This marks the beginning of secondary glaucoma. The tension mounts gradually and may progress to absolute glaucoma with complete closure of the chamber angle. The etiology of the malady is unknown, and many theories have been postulated as to the mechanism of each phase in its development; several of the numerous theories are discussed.

A 38-year-old woman is described who had noted some change in her pupil ten years ago. The original impression was that of polycoria, but the development of glaucoma prompted a more thorough investigation at which time the characteristic changes confirmed the diagnosis of essential atrophy of the iris. There were visual field defects, typical of glaucoma. Cyclodialysis, combined with sclerectomy, normalized the tension with a final corrected vision of 1.0 (53 references)

James W. Brennan.

Stanworth, A. **Rheumatism and uveitis.** Tr. Ophth. Soc. U. Kingdom **76**:287-296, 1956.

Of 237 consecutive cases of uveitis, 40 occurred in patients with significant rheumatic disease, which is mainly associated with anterior uveitis of nongranulomatous or doubtful types. No association was found between uveitis and osteoarthritis or classical rheumatoid arthritis.

Nongranulomatous anterior uveitis was

associated with spinal arthritis and Reiter's disease. Ten percent of patients with rheumatoid arthritis had dry eyes and the author believes that there was an increased incidence of rheumatoid arthritis in patients with keratitis sicca. (2 figures, 5 references) Beulah Cushman.

9

GLAUCOMA AND OCULAR TENSION

Agarwal, L. P. and Malik, S. R. K. **Pre- and post-operative acetazoleamide (Diamox) in glaucoma surgery.** Brit. J. Ophth. **41**:613-615, Oct., 1957.

Fifty patients with glaucoma were hospitalized and given single oral doses of acetazoleamide of 500 to 1000 mg. daily for two to three days. One hour before surgery the patient was given 500 mg. of the same drug but 250 mg. of this was of the delayed-action type. This same medication was continued for one week postoperatively. The surgical procedure was a post-placed valvular iridencleisis. Fifty additional patients with glaucoma were used as control inasmuch as they received no acetazoleamide except in those cases in which no filling of the chamber was noted by the fifth postoperative day. In the control group the incidence of flat chamber up to the fifth day was 42 percent whereas in the group under acetazoleamide it was six percent. The type of bleb in the control series was definitely larger and more cystic than in the medicated group, but both types seemed to function adequately. Pre- and postoperative use of acetazoleamide is suggested as a routine procedure. (3 tables, 9 references) Lawrence L. Garner.

Gloster, J. and Greaves, D. P. **Effect of diencephalic stimulation upon intraocular pressure.** Brit. J. Ophth. **41**:513-532, Sept., 1957.

Male cats in stereotaxic apparatus were used and under direct electrode stimulation of the diencephalon, simultaneous recordings were made of the pupillary responses, effects on the nictitating membrane, the blood pressure changes and the intraocular pressure changes. The points of stimulation on the brain tissue followed various patterns which were correlated with the patterns of response.

Dilatations of the pupils occurred over a wide area of stimulation and always occurred in both eyes simultaneously while constrictions of the pupils rarely were noted. Movements of the nictitating membranes were small and of no significance. Blood pressure changes varied considerably and occurred both with and without intraocular pressure changes. About half the stimulations resulted in no change in either blood or ocular pressures but in most instances in which a change was noted, the change was found in both. It was felt that often a fall in intraocular pressure followed and was caused by a reduction in the ocular blood supply and this fact may well be a key in solving the association between the diencephalon and glaucoma. More investigative studies of this problem are urged. (12 figures, 1 table, 16 references) Morris Kaplan.

Higgitt, A. C. **Secondary glaucoma.** Tr. Ophth. Soc. U. Kingdom **76**:73-82, 1956.

The author reports that about seven percent of the patients in the glaucoma clinic had secondary glaucoma and if a quarter of the angle remained open the tension could be controlled by miotics. The common finding in the different glaucomas was the raised ocular tension due to obstructed aqueous outflow. Gonioscopy was the chief method of differentiating the secondary glaucomas. (3 figures) Beulah Cushman.

Huggert, A. **An experiment in determining the pore-size distribution curve to the filtration angle of the eye.** Acta ophth. **35**:11-19, 1957.

This is the first part of the report, and

it deals with evaluating the curves obtained with perfusion of isobutyl alcohol. The literature concerning perfusion experiments with physiologic saline or aqueous humour is reviewed. It is shown that in perfusion with isobutyl alcohol long periods of time are necessary to obtain constant values, especially at low pressure levels. Mechanical deformation of the filter is less with isobutyl alcohol than with aqueous or physiologic saline. Age of the animal has to be taken into consideration in perfusion experiments with all three fluids. (5 graphs)

Ray K. Daily.

Huggert, A. **Experimentally produced obstruction to the outflow of aqueous humour.** Acta ophth. **35**:1-11, 1957.

Three methods of creating obstruction to outflow were used, 1. diathermy cauterization of the episcleral aqueous veins near the limbus; 2. ligation of the vessel trunks at the posterior pole of the globe; and 3. injection of a suspension of particles into the anterior chamber. The appearance of fluorescein in the anterior chamber after its intravenous injection was the criterion for the evaluation of the variations in the inflow. The data show that all types of blockage produced a rise in intraocular pressure and impeded the inflow. The elevated ocular tension lasted longer in obstruction caused by ligating at the posterior pole and by blockage of the filtration angle. Some of the ligated eyes remained at high pressure levels for longer than six months. The eyes in which the episcleral aqueous veins were cauterized showed a great capacity for compensation; new paths for outflow, in the form of new superficial channels, and increased flow through deeply seated channels developed rapidly. The role of an adjustment in the inflow in the restoration of normal intraocular pressure was not definitely determined in this investigation; it appears that such an adjustment must have taken place, as the compensation in the outflow channels alone could not have been sufficient. The experiments with blockage of the filtration angle demonstrated that particles under 0.5 microns in diameter had no blocking effect. Particles of 7.5 microns in diameter created blockage. (2 graphs, 1 table, 12 references)

Ray K. Daily.

Keerl, Gerhard. **The atypical glaucomatous field defect.** Klin. Monatsbl. f. Augenh. **131**:505-513, 1957.

In five patients with glaucoma and axial myopia the defect in the visual field started temporally and extended fan-like toward the blind spot. It is assumed that the different structure of the myopic disc exposes first the nasal nerve fibers to the effects of an increased intraocular pressure. (5 figures, 27 references)

Frederick C. Blodi.

Lavergne, G., Prijot, E. and Weekers, R. **Recent advances in tonometry.** Arch. d'opht. **17**:256-270, 1957.

The authors review the present status of tonometry so far as the methods of indentation of the cornea and aplanation of the cornea are concerned. They discuss in detail tonometry with the Schiøtz tonometer and refer particularly to the role of scleral rigidity in measurements. They urge the use of the Friedenwald calibration tables (1955), and conclude that the normal ocular tension in subjects lying prone averages 17 mm. Hg, with a normal range of variation from 8.5 to 25.2 mm. Hg. With the aplanation technique the average normal pressure is 15.70 mm. Hg. (11 figures, 2 tables, 7 references)

P. Thygeson.

Law, Frank W. **Iridencleisis in buphthalmia.** A.M.A. Arch. Ophth. **58**:357-362, Sept., 1957.

The author reports eleven cases of buphthalmus in which iridencleisis was

done. He believes that his results are as good as those of the newer goniotomy procedures. He says that digital examination is more to be relied upon than tonometry in estimating tension. (2 figures, 28 references) G. S. Tyner.

Leopold, I. H., Gold, P. and Gold, D. **Use of a thiophosphinyl quaternary compound (217-MI) in treatment of glaucoma.** A.M.A. Arch. Ophth. **58**:362-366, Sept., 1957.

The authors report on a new anticholinesterase drug for the control of intraocular pressure. The advantages claimed are 1. long action, 2. stability for long periods when kept cool, 3. control of cases not previously controllable, 4. freedom from dermatitis and conjunctivitis, and 5. lack of systemic effects. It does cause some blurring and discomfort. (4 tables and 8 references) G. S. Tyner.

Seifert, Eberhard. **A fixation device for the Schiøtz tonometer.** Klin. Monatsbl. f. Augenh. **131**:405-406, 1957.

The tonometer is attached to a trial frame. It can so be held in position on the eye during tonography. (6 figures)
Frederick C. Blodi.

Unger, Lothar. **Bandaging of the eyes during the night as a provocative test for glaucoma.** Klin. Monatsbl. f. Augenh. **131**:376-385, 1957.

During the night the patient's eyes are covered with a black cloth which is secured by a bandage. The bandage is removed in the morning and the tension recorded immediately. A rise of over 7 mm. or an endpoint of over 30 mm. Hg. is considered pathologic. 200 normal eyes were tested and none showed an increase of over 5 mm. Hg. Six patients are described in whom this test was positive while other provocative measures were negative. The author believes that the disturbed sleep with increased sympathicotony causes the increase in pressure. (6 figures, 39 references) Frederick C. Blodi.

Whiting, M. H. **Herbert's sclerotomy.** Tr. Ophth. Soc. U. Kingdom **76**:283-286, 1956.

The author presented a patient with a gonioscopic puncture with a satisfactory filtrating scar and ten years later with normalized tension. (1 figure)
Beulah Cushman.

10
CRYSTALLINE LENS

Atkinson, Walter S. **Pre- and postoperative care of cataract patients.** Geriatrics **12**:571-580, Oct., 1957.

In this well written and informative paper the author describes in detail such important maneuvers in pre- and postoperative care of cataract patients as preanesthetic preparation, cultures, preparation of the operative field, sedation and analgesia, anesthesia, akinesis and the use of curare. (10 figures, 9 references)
Irwin E. Gaynon.

Costenbader, F. D. and Albert, D. G. **Conservatism in the management of congenital cataract.** A.M.A. Arch. **58**:426-430, Sept., 1957.

The authors agree with former writers on the poor outlook after surgery for congenital cataract. They advise against surgery for unilateral cataract. They discuss the advantage of optical iridectomy and prolonged use of mydriatics. (7 references) G. S. Tyner.

Hambresin, L. and Ledere, J. **Cataract operations in diabetics.** Bull. Soc. belge d'opht. **115**:240-257, 1957.

Precautionary measures in pre- and postoperative treatment of cataract in diabetics and the special techniques of the surgery, especially the performance of simultaneous or preparatory iridectomy are discussed. The authors operated

with good results on six diabetics with cataract, aged 32 to 65 years. Chabaier's statement "a diabetic, well prepared, has at present as good a chance for successful cataract operation as anybody else" therefore seems to be confirmed.

Alice R. Deutsch.

King, J. H., Jr. and Skeehan, R. A., Jr. **Acrylic lenses in the anterior chamber.** A.M.A. Arch. Ophth. **58**:392-395, Sept., 1957.

The authors review the literature and report on their experiments with animals in placing acrylic lenses in the anterior chamber with only minimal ocular complication. (5 figures, 6 references)

G. S. Tyner.

Lee, P. and Trotter, R. R. **Tonographic and gonioscopic studies before and after cataract extraction.** A.M.A. Arch. Ophth. **58**:407-416, Sept., 1957.

After studying 142 eyes with cataracts, the authors conclude that after cataract surgery the facility of outflow of aqueous may be temporarily decreased, unchanged, or increased. Eyes with exfoliation of the lens capsule or open-angle glaucoma have an unchanged facility of outflow and intraocular pressure after cataract extraction. Early gonioscopy should be done after a flat anterior chamber, for miotics may break some of the peripheral anterior synechias. (4 figures, 3 graphs, 3 tables, 4 references) G. S. Tyner.

Miller, J. E., Keskey, G. R. and Becker, B. **Cataract extraction and aqueous outflow.** A.M.A. Arch. Ophth. **58**:401-406, Sept., 1957.

In order to get information on the glaucoma following cataract extraction, 111 patients were subjected to repeated tonography and gonioscopy before and after cataract surgery. The authors conclude that even in uncomplicated cases there is some damage to the aqueous outflow; complications increase the effect. Rupture of the lens capsule at the time of surgery resulted in a 30-percent incidence of glaucoma, and accounted for two-thirds of the secondary glaucoma in this series. Cataract extraction is followed by a hyposecretion of aqueous humor for a considerable period. (1 table, 7 references)

G. S. Tyner.

Reese, W. S. and Hamdi, T. N. **Five years' experience with the Ridley operation.** A.M.A. Arch. Ophth. **58**:389-391, Sept., 1957.

The authors report their experiences with 115 cases over a five year period. They stress that they discuss frankly with the patients the disadvantages of the technique and the fact that the procedure is designed primarily for unilateral cataracts. They discourage the operation in persons with bilateral cataract. The early reactions and the extra care needed are mentioned, but the report is in general quite favorable. (4 graphs, 3 references)

G. S. Tyner.

11

RETINA AND VITREOUS

Adams, Samuel T. **Pars plana cysts.** A.M.A. Arch. Ophth. **58**:328-330, Sept., 1957.

While using the Schepens technique one sometimes sees pars plana cysts in the extreme periphery of the fundus. The author describes the gross appearance and the histologic picture. He believes that they are of no clinical importance. (8 figures and 11 references) G. S. Tyner.

Bialasiewicz, A. **Priscoline in the treatment of central serous retinopathy.** Klin. Monatsbl. f. Augenh. **131**:536-537, 1957.

Priscoline was injected intramuscularly in nine patients; the results were promising. Frederick C. Blodi.

Fison, Lorimer. **Planned treatment of retinal detachments.** Tr. Ophth. Soc. U. Kingdom **76**:259-266, 1956.

The author states that the first essentials are the complete examination of each fundus and the drawing of an accurate map by the surgeon to show the principal vessels and holes so that the best treatment can be planned beforehand to replace the retina and seal off any holes and thin areas in it. Binocular indirect ophthalmoscopy with a +20D. aplanatic lens offers the best method of such fundus examinations. A careful history should note especially the presence and direction of flashes of light and the time of the sudden increase of floaters. The periphery of the fundus is examined with the help of a specially shaped rod mounted on a thimble which is worn on the middle finger of the left hand and with which each quadrant of the globe is indented for inspection.

The patient is then put into bed in a position in which the retina is most apt to settle back against the wall of the globe by gravity. This may require several days and when the maximum effect has been obtained the site of the retinal holes should again be verified.

Surface diathermy under direct observation is best suited to a simple hole or an isolated group of holes in the retina. Lamellar sclerectomy giving a long area of reaction will provide a means of holding an area of retina with several holes in it or holes with adjacent thin retina. Surface diathermy must be applied to the holes before the sclerectomy is made. After the main operation to produce the sticky patch of choroidoretinal exudate, the subretinal fluid is evacuated by scleral puncture. Beulah Cushman.

Frayer, William C. **Improvement in hypertensive retinopathy following adrenal resection and sympathectomy.** A.M.A. Arch. Ophth. **58**:331-336, Sept., 1957.

The author describes the retinal changes observed in 111 severely hypertensive patients after adrenalectomy and sympathectomy. Recession of the retinopathy was noted in 78 percent, the hemorrhages, exudates, and edema often disappearing rapidly. This improvement may occur without improvement in blood pressure and is probably the result of diminished peripheral resistance. (6 tables, 18 references) G. S. Tyner.

Kittel, V. **The diagnostic importance of the peripheral arterial pulse in the retina.** Klin. Monatsbl. f. Augenh. **131**:500-505, 1957.

The serpentine type of arterial pulsation occurs quite frequently in normal eyes. It is better visible in the nondilated pupil and depends to a large extent on the character of the retinal reflexes. (1 figure, 12 references) Frederick C. Blodi.

Ohrt, Vagn. **Gyrate atrophy of choroid and retina and inverse retinitis pigmentosa in the same patient.** Acta ophth. **35**:26-31, 1957.

The patient, a man 20 years of age, had a fundus picture characterized by moderate pallor of the optic discs, narrow arteries with normal veins, irregular pigmentary changes at the posterior poles, exposure of the choroidal vessels in three quadrants of the fundus, and atrophic patches bordered by pigment. The patient always had night blindness, and defective vision from childhood. The diagnosis was inverse retinitis pigmentosa and gyrate atrophy of the choroid. It is believed to be the first reported case of the combination of these two diseases. After a brief review of the literature on retinal dystrophies the author agrees with Sorsby that the various clinical types of retinal dystrophy are not rigid entities, but local-

ized expressions of one fundamental disturbance. (12 figures, 7 references)

Ray K. Daily.

Schlitter, K. **The question of retrolental fibroplasia.** Klin. Monatsbl. f. Augenh. **131**:544-547, 1957.

The author describes a nearly 7-year-old girl (birth weight 2950 gm.) who had retrolental opacities not unlike those seen in retrolental fibroplasia. The girl had never had supplementary oxygen. (19 references) Frederick C. Blodi.

Schmoeger, Elizabeth. **The prognostic value of the electroretinogram in retinal detachment.** Klin. Monatsbl. f. Augenh. **131**:335-343, 1957.

Four patients had an ERG before and after the operation. The ERG was reduced or negative in all patients and yet the postoperative visual result was good or excellent. A reduced ERG in retinal detachment does not have prognostic value. (5 figures, 10 references)

Frederick C. Blodi.

Stephenson, R. W. **Anti-coagulant therapy in retinal vein thrombosis.** Tr. Ophth. Soc. U. Kingdom **76**:253-258, 1956.

Three main sites of venous block are at the level of the lamina cribrosa, at the arteriovenous crossings and at the point where a branch joins another vein. The author found that nothing could be achieved by treating patients who have a vein completely blocked unless the block appeared to be extending into a larger vein. Restoration of vision and a normal fundus can be achieved more quickly by treatment of an incomplete block and treatment was continued for three or four months. No complicating hemorrhages occurred in the patients treated.

Beulah Cushman.

Wadsworth, Joseph A. C. **The vitreous.** A.M.A. Arch. Ophth. **58**:455-464, Sept., 1957.

Wadsworth claims that gross pathologic changes in the vitreous can be correlated and confirmed by microscopic and clinical findings. Many pictures previously described as artefacts are actual pathologic entities in the vitreous. The relationship of detachment of the retina to changes in the vitreous is discussed. (42 figures, 10 in color, 36 references)

G. S. Tyner.

Wolter, R. J. and Jampel, R. S. **Retinal folds produced by pressure of a tumor on the globe.** Klin. Monatsbl. f. Augenh. **131**:433-438, 1957.

This eye had to be enucleated because of an osteosarcoma of the maxilla invading the orbit. Two types of folds could be seen on histologic section. One involved the entire thickness of the retina and the inner layers showed a more pronounced folding. In the second type only the internal limiting membrane and the nerve fiber layer were affected. (3 figures, 6 references) Frederick C. Blodi.

12

OPTIC NERVE AND CHIASM

François, J., Verriest, G. and Baron, A. **Pseudo-papillitis.** Acta ophth. **35**:32-50, 1957.

By vascular pseudopapillitis the authors signify an optic neuritis in which no generalized or localized focus of infection can be found, and the presence of vascular disturbances can be demonstrated. Seven case reports illustrate the course of such lesions in arteriosclerosis and temporal arteritis. The literature on its possible occurrence in diabetes, periarteritis nodosa, Raynaud's disease, and polycythemia is reviewed. The authors consider therapy in such cases generally ineffective. The prodromal signs of the lesion are periorbital pains and transient obscurations of vision. The lesion sets in suddenly with a significant loss of vision, and the fundus shows peripapillary edema and sometimes

hemorrhage and exudate. The retinal arteries are narrow and the veins dilated. The process proceeds to a secondary optic atrophy, which may resemble a primary or postneuritic in type. In three cases examined with the electroretinograph and the critical fusion test the data were identical in the involved and uninvolved eye. All seven patients had electroencephalographic and electrocardiographic disturbances indicating an inadequate circulation in the heart and brain. The authors refer to the viewpoint held by some investigators, who maintain that all vascular pseudopapillitis is caused by a granulomatous arteritis. The anatomo-pathologic examination of the temporal artery in two of the authors' cases does not support this contention. (11 figures, 43 references) Ray K. Daily.

13

NEURO-OPHTHALMOLOGY

Alexander, G. L. **Diagnostic value of colour fields in neurosurgery.** Tr. Ophth. Soc. U. Kingdom **76**:235-244, 1956.

The author found that the inconsistency in visual fields with the smallest white test objects in individual patients disappeared when a larger colored object was used. He uses a 2 mm. target of as pure a red as possible (Dennison crepe paper, flame-red) on one side and white on the other and warns the patient that it may appear gray or orange before it turns red and to say red when he sees it as red. He favors artificial lighting in a room to make field taking on a Bjerrum screen as convenient and constant as possible. (9 figures) Beulah Cushman

Busch, Karl Theodor. **The early diagnosis of pinealomas.** Klin. Monatsbl. f. Augenh. **131**:304-313, 1957.

This report is based on the experience in eight patients. The earliest symptoms were those of increased intracranial pressure and papilledema. Anisocoria and pupillary disturbances followed. Diplopia is frequent and usually due to damage to the fourth or sixth cranial nerve. Deafness, cerebellar symptoms and gaze palsy upward are late symptoms. Three of the eight patients had a bitemporal hemianopia, one had a homonymous hemianopia and three patients were too sick to have a field examination. (4 figures, 17 references) Frederick C. Blodi.

Dreyer, Viggo. **Choked discs of unknown origin: 16 cases in which intracranial expansive processes could be excluded as etiological factors.** Acta Psychiat: & Neurol. Scandinav. **32**:20-36, 1957.

The author provides an excellent discussion on the definition, meaning, and significance of choked disc. Of the 16 abnormal discs, five were physiologic variants, three pseudoneuritis, one drusen of the disc, and one hyperemic disc; the other 11 were classified as true choked disc because the fundus picture changed during the period of observation. In nine patients the protrusion subsided, and in two it remained stationary. All patients were under the age of 50 years, and 12 were female.

The chief causes of the true choked discs which were not found to be cryptogenic, were post-traumatic arachnoiditis in six or seven cases and aseptic sinus thrombosis in three cases. In most of these cases the intraventricular pressure was normal, or near normal. The prognosis in this group is good; vision was preserved in all and a lasting visual field defect occurred in just one. (2 tables, 16 references) Harry Horwich

Minton, Joseph. **A case of left herpes zoster ophthalmicus followed by virus encephalitis with right-sided anesthesia, paresthesia and hemiplegia.** Tr. Ophth. Soc. U. Kingdom **76**:227-232, 1956.

The author describes a patient, 47 years of age, who had severe left herpes zoster

ophthalmicus in which the cornea and uvea were involved. Six weeks later he developed anesthesia and paresthesia of the right side of the body with weakness of the right leg and arm. The neurologic changes were probably due to a spread of the infection from the left Gasserian ganglion to the left cortex. Usually virus encephalitis is fatal, but this patient fully recovered. Ten months after the onset of of the herpes zoster ophthalmicus the cornea was grossly thickened and vascularized and vision was reduced to shadows of large objects. Systemic treatment with ACTH and local treatment with hydrocortisone has resulted in the clearing of the cornea and abatement of the inflammatory changes in the iris. The vision improved to 6/9. (5 figures, 20 references) Beulah Cushman.

Walsh, Frank B. **Third nerve regeneration.** Brit. J. Ophth. **41**:577-598, Oct., 1957.

Third-nerve paralysis is followed in some instances by an aberrant type of regeneration resulting in some bizarre ocular and lid movements due to the misdirected growth of the nerve fibers. Once this aberrant type of regeneration takes place no spontaneous improvement can be anticipated. The greatest percentage of occurrence can be expected in third-nerve paralysis associated with trauma or aneurysm, while none has been noted in third-nerve paralysis associated with diabetes or ophthalmoplegic migraine. It may occasionally be seen with third-nerve paralysis due to tumor or inflammation. Surgery for persistent ptosis is possible. (14 figures, 37 references)

Lawrence L. Garner.

14

EYEBALL, ORBIT, SINUSES

Bartolozzi, R. **Cranio-orbital processes.** Arch. Soc. oftal. hispano-am. **17**:921-1099, Aug., 1957.

This comprehensive monograph covers all lesions of the skull and facial bones, which may involve the orbit. (47 figures, 541 references) Ray K. Daily.

Bertelsen, T. I. and Petersen, C. **Orbital pneumography.** Acta Radiol. **47**: 426-432, June, 1957.

The techniques for injection of air beneath Tenon's capsule and behind the globe are described in detailed. Contraindications are infection and suspected rupture of the globe. Tomography in two planes at intervals of 0.5 cm. is recommended. Eight patients were examined by this method, in whom all other types of investigation had failed to reveal the cause of unilateral exophthalmos. Three of them are described; one showed no abnormality, and in two there was a mass of soft tissue. (9 figures, 14 references)

Harry Horwich.

Clausen, A. **The cavernous hemangioma of the orbit.** Klin. Monatsbl. f. Augenh. **131**:401-404, 1957.

In a 71-year-old woman with unilateral exophthalmus of only two weeks duration the superior orbital fissure was enlarged. Histologic examination revealed a hemangioma with numerous lymph follicles. (3 figures, 17 references)

Frederick C. Blodi.

Gordon, S. **Malar fracture, intra-orbital hemorrhage during open reduction.** Plast. & Reconstruct. Surg. **20**:65-66, July, 1957.

A 63-year-old man is described whose lateral orbital wall was crushed medially, resulting in subconjunctival hemorrhage in the lateral half of the right eye, numbness over the distribution of the right infraorbital nerve, and bruising of both right eyelids. At surgery, the fracture was reduced by means of the Bristow elevator, but at the completion of surgery the eye was proptosed and the lids were swollen and tense. When tension continued to mount within the orbit, a transverse inci-

sion was made at the junction of the eyelid and cheek skin under the mid-third of the palpebral fissure, and the point of a small blunt curved scissors was pushed through the incision along the orbital floor and then separated. This was followed by marked arterial hemorrhage, the orbital swelling decreased and the eye returned to its proper position. The vision of the eye returned to normal. Alston Callahan.

Hudson, J. R. **Present status of orbital implants and prosthesis.** Tr. Ophth. Soc. U. Kingdom **76**:245-252, 1956.

The author published a paper six years previously describing a modified technique for introduction of the Cutler implant, and he now says that whatever the difficulties of the integrated implant, it does produce by far the best cosmetic result. The objectives to be achieved by operation are a wide range of movement, instantaneous movement over short ranges, the absence of sinking in of the upper lid and absence of sagging of the lower lid, and permanence.

After disappointments with the partly buried implants he has used the Castroviejo implant since 1952 and none have been extruded. The Castroviejo implant resembles the Allen implant, and Roper-Hall has modified one by the introduction of a magnet. The Arruga implant had been successful as reported by Count Arruga in the British Journal of Ophthalmology. (1 figure) Beulah Cushman.

Lemoine, Albert N., Jr. **The orbit.** A.M.A. Arch. Ophth. **58**:468-474, Sept., 1957.

The lesions of the orbit are well reviewed and emphasis is given to a paper by Jackson on conservation of the eyeball in the treatment of certain tumors of the orbit. (52 references) G. S. Tyner.

Mustarde, J. C. **Reconstruction of the grossly contracted socket.** Tr. Ophth. Soc. U. Kingdom. **76**:297-309, 1956.

The problems of contracted sockets cannot be underrated and the author gives some hints for enlarging them and for the care necessary to prevent entropion and ectropion. (8 figures)

Beulah Cushman.

Nover, A., Josten, K. and Zielinski, H. W. **The range through which an eye can be pressed back into its orbit.** Arch. f. Ophth. **159**:233-245, 1957.

A statistical evaluation of a number of factors was based on concurrent measurement of 727 eyes with the piezometer of Jaeger and the exophthalmometer. The amount which an eye could be pushed back was less than normal in eyes with myopia or ocular tension greater than 60 mm. Hg and greater than normal with hyperopia and phthisis bulbi. The authors also call attention to a relationship between exophthalmometry and piezometry. (7 figures, 22 references)

F. H. Haessler.

Spanyol, V. **The treatment of orbital hemangiomas by aspiration.** Klin. Monatsbl. f. Augenh. **131**:448-451, 1957.

In two children puncture and, when necessary, repeated aspirations of this orbital tumor led to a considerable reduction of the exophthalmus. (1 reference)

Frederick C. Blodi.

15

EYELIDS, LACRIMAL APPARATUS

Bennett, J. E. and Bailey, A. L. **A surgical approach to total xerophthalmia.** A.M.A. Arch. Ophth. **58**:367-371, Sept., 1957.

The successful transplantation of the parotid duct into the conjunctival sac is reported in four dogs. The authors quote the favorable results in man reported in the Chinese and Russian literature. (4 figures, 1 table, 11 references) G. S. Tyner.

Calmettes, L. Deodati, F. and Daraux, H. **Congenital dyskeratosis with atresia of the puncta.** Arch. d'opht. **17**:250-255, 1957.

The authors add a case description to the ten already published of this rare disease, congenital dyskeratosis, also known as the disease of Zinsser, Engman, and Cole, the dermatologists who originally described it. The essential features consist of a reticular pigmentation of the skin, a dystrophy of the nails, and mucocutaneous disturbances of the various orifices of the body. The authors note that all cases have been in males and that in three instances the affection has occurred in brothers. There has been no other evidence of an heredity factor, however. The age of onset has been between 5 and 12 years, infancy in all cases having been normal. They note that dystrophy of the nails has been an invariable feature, and that leucoplasia and atresia of the puncta have occurred regularly. Among the eye signs noted in addition to those affecting the lacrimal apparatus have been conjunctival vesicles and bullae and a special pigmentation of the fundus. The authors note the resemblance of congenital dyskeratosis to Darier's disease, to congenital poikilodermia of Thomson, and to the atrophic vascular poikilodermia of Jacobi. (6 figures, 21 references) P. Thygeson.

Guenther, G. **Reoperation after dacryocystorhinostomy.** Klin. Monatsbl. f. Augenh. **131**:343-350, 1957.

Six operations are reported. The cause of failure was too small a bony window in five patients and a faulty position of the window in the sixth. (12 references) Frederick C. Blodi.

Valentin-Gamazo, Ignacio. **Blepharoplasty for carcinoma of the lower lid.** Arch. Soc. oftal. hispano-am. **17**:1112-1121, Sept., 1957.

The author reports a case of carcinoma of the inner angle of the right lower lid in a man 50 years old. The tumor was extirpated and the defect repaired by utilizing a sliding flap from the cheek. The author urges that the clinical classification of these neoplasms should be based not on their histologic morphology, but on the extent of their invasion of the tissues. He is in favor of adopting Kreibig's classification. He urges that surgery be accepted as the procedure of choice, with irradiation as a postoperative adjunct, when indicated. He also urges a systematic follow-up for life of all patients treated for malignancy of the lids. (6 figures, 13 references) Ray K. Daily.

Weber, Joachim. **A modification of the Friedenwald-Guyton ptosis operation.** Klin. Monatsbl. f. Augenh. **131**:407-409, 1957.

In order to avoid the nasal notch a second suture is added. This suture goes subcutaneously from the center of the lid margin to the center of the brow. It is removed after seven days. (5 figures, 5 references) Frederick C. Blodi.

16
TUMORS

Gasteiger, H. and Nonnenmacher, H. **Misdiagnosis of malignant intraocular tumors.** Klin. Monatsbl. f. Augenh. **131**: 293-304, 1957.

Three patients with choroidal melanoma are discussed, in whom the diagnosis could only be made after prolonged observation. Transillumination was usually misleading. In one patient the subretinal fluid was aspirated and its histologic examination did not reveal any tumor cells. Traumatic hemorrhage, Coats' disease and inflammatory retinitis were the original diagnoses.

In two children with retinoblastoma (eight and nine years old) enucleation was done only after months of evaluation. In the first child a diagnosis as Coats'

disease was made though two of her uncles and all her four siblings had retinoblastoma. The other child presented the clinical picture of secondary glaucoma. (8 figures, 1 reference)

Frederick C. Blodi.

Pfanz, Waldemar. **Lymphatic tumors of the eye.** Klin. Monatsbl. f. Augenh. **131**:439-447, 1957.

Seven patients are described. In four of them the tumor was orbital. In one patient the tumor arose in the lid and the two other patients had conjunctival tumors. In two patients with orbital reticulum-cell lymphoma exenteration was done. One patient died, the others improved on X-radiation. (6 figures, 19 references) Frederick C. Blodi.

17

INJURIES

Cunha, S. L. **Ocular burns from pathogenic acids and alkalies and their treatment.** Arq. brasil. de oftal. **19**:276-294, 1956.

The importance of chemical burns of the eye has been recognized in ophthalmic literature since 1813. Credit goes to George Beer as one of the foremost authors on the subject. It has been recognized that alkalies, of the two, produce the most serious lesions in the eye. Burns occur often in peacetime, but are especially frequent in wartime, because of the use of chemicals in the manufacture of munitions and as weapons.

The corneal epithelium acts as a barrier to the deep penetration of acids. After destruction of the epithelium, the acid penetrates the stroma, forming an insoluble protein. The chemical action of an acid is instantaneous and the protein affinity of its anion determines the degree of resultant injury. Among those with a high affinity for protein are trichloracetic, sulfosalycilic, picric, and tannic acids. The destructive process occurs in two stages, one of coagulation (denaturation) of the protein, and the other the formation of a coagulum by flocculation of the denatured molecules. The former is a chemical reaction of hydrolysis and is reversible, while the latter is a physical reaction and is irreversible. Vascular response to acids is intense, but only few inflammatory cells appear. Late relapses are rare.

All alkali burns have common features in that the lesion tends to progress in severity and late complications are frequent. Saponification of the fat in the corneal epithelium permits deep penetration of the chemical, with the formation of soluble proteins which do not impede further penetration of the caustic. The endothelium may be destroyed and the anterior chamber invaded with subsequent iridocyclitis, capsular cataract and secondary glaucoma.

It is postulated that there are three actions of an alkali upon the eye—production of heat, dehydration by a hygroscopic effect and a direct action upon corneal elements. In the latter category are loss of transparency of collagen, loss of mucoid, disappearance of cellular elements, formation of toxic hydrolysates, and vascular necrosis of limbal vessels. Other important factors in determining the degree of injury are the duration of exposure or contact, the pH of the caustic and the velocity of penetration through the tissues.

Immediate treatment consists in copious irrigation with running water. Neutralizing agents are condemned in most instances. Solid particles (lime) should be removed. The eye should be examined by a specialist, with fluorescein instilled and followed by irrigation with saline solution. The conjunctival fornices should be inspected, and any adherent particles which cannot be removed should be covered with oil or glycerin. The role of paracentesis in caustic burns is mentioned

as being helpful when the penetration has been rapid. Contaminated or necrotic tissue is excised. Toxic hydrolysates are removed from the subconjunctival space by excising necrotic tissue and covering the defect with a graft which may be labial mucosa, amniotic membrane, or animal conjunctiva.

Subsequent therapy consists of the use of antibiotics to combat secondary infection, mydriatics, medication to maintain vascular efficiency (vasodilators), cortisone and ACTH. The administration of sulfhydryl and antihistamines has been recommended by several authors. Symblepharon should be avoided as much as possible by breaking adhesions with a glass rod, the insertion of conformers or contact lenses and the local administration of cortisone. Corneal opacities have been treated with a variety of agents, and may require keratoplasty. Vascularity may be treated by diathermy application or Beta radiation, and severe glaucoma may require enucleation for relief of pain. (86 references) James W. Brennan.

Landolt, E. **A case of fat embolism in the eye.** Klin. Monatsbl. f. Augenh. **131**: 538-540, 1957.

A 29-year-old man died nine days after a serious accident. Retinal hemorrhages and white patches were seen ophthalmoscopically one day before his death. Fat was found in the retina (flat sections), choroid, sclera, ciliary body and optic nerve. (3 figures, 4 references)

Frederick C. Blodi.

May, W. **Intra-ocular fragment of aluminum alloy tolerated for 12 years.** Brit. J. Ophth. **41**:574-575, Sept., 1957.

In 1944 a young man was struck in the face by a blast of metallic particles; cataract in one eye resulted in loss of vision after cataract extraction. The eye remained quiet for 12 years and was seen again for an unrelated complaint. A foreign body was found in the anterior chamber, resting on the iris. It was removed by keratome incision and found to be an alloy of aluminum which had apparently been well tolerated through the years. (1 figure, 1 reference) Morris Kaplan.

Neubauer, Hellmut. **Traumatic chorioretinopathy with sector-shaped field defect.** Klin. Monatsbl. f. Augenh. **131**:487-500, 1957.

Three patients are described in whom a blunt injury caused a choroidal atrophy above the disc and a defect in the lower field. It is assumed that this disturbance is due to a lesion in one of the posterior ciliary arteries which may be damaged when the eye is forcefully elevated (Bell's phenomenon) and struck from in front. (5 figures, 12 references)

Frederick C. Blodi.

Planten, J. T. **Intraocular foreign body.** Ophthalmologica **133**:432-433, June, 1957.

In a case of perforating injury and retention of a piece of steel in the vitreous, the gradual development of siderosis is described in detail. Presented at the 136th meeting of the Dutch Ophthalmological Society the case gave rise to an extensive discussion which brought out the advantages of small, easily manageable magnets over the giant magnet and the more favorable prognosis in cases of retained foreign bodies consisting of stainless steel.

Peter C. Kronfeld.

18

SYSTEMIC DISEASE AND PARASITES

Alaerts, L. and Sloane, J. **Ocular complications of diabetes.** Bull. Soc. belge d'opht. **115**:162-183, 1957.

In this instructive report the ocular complications of diabetes are divided into two groups. The pathologic changes of the aqueous, the pigment epithelium of the iris, the lens and various neuropathies

are ascribed to toxic causes, and abnormalities of the iris stroma and the retina are identified with vascular disease. The variations in the composition of the aqueous, the clinical appearance of pigment scattering and the difficulties in the evaluation of lens opacities and refractive changes are described. It is emphasized that diseases of the optic nerve and the cranial nerves are relatively rare and not especially characteristic in their symptomatology.

Diabetic iridopathy, iritis in diabetes, diabetic rubeosis of the iris and posthemorrhagic rubeosis and the criteria of importance in their differential diagnosis are discussed. The vascular phenomena of the retina are evaluated in relation to length of illness, simultaneous hypertension, glomerulosclerosis and sudden or gradual variations in the individual response to insulin. The retinal lesions are often independant of the stage of the diabetes; nevertheless minimal abnormalities undoubtedly are the first signs of a threatening decompensation. Aneurysms and small hard exudates are thought to be independent of hypertension and general sclerosis. They may follow any local or general primary or secondary capillaropathy. (17 references)

Alice R. Deutsch.

Appelmans, M., Doyen and Walgravens. **Insulin-resistant para-diabetes associated with acromegaly, cataract and retinitis.** Bull. Soc. belge d'opht. **115**:260-267, 1957.

The ocular complications of paradiabetes and diabetes mellitus were compared while studying two patients with acromegaly, insulin-resistant hyperglycemia, cataract and retinitis. Both patients had had X-ray treatment during the early years of the disease because of severe headaches. The sella was found to be enlarged. Nevertheless the chiasm was not affected. Glucose tolerance was decreased in both patients. The hyperglycemia did not improve with the administration of insulin. The lens opacities were uncharacteristic posterior cortical cupuliform cataracts and could have been the result of the metabolic disturbances or the X-ray treatment or both. The retinal changes resembled diabetic retinopathy. One of the patients had an enlargement of the lacrimal and salivary glands, the other had a hypertrophy of the thyroid with exophthalmos. The prognosis of paradiabetes is in general poor for preservation of vision and duration of life. Only a few paradiabetics lived to be 60 years old whereas diabetics often reach an age of 70 years or more.

Acromegaly was described for the first time by Pierre Marie in 1886 and the abnormality was ascribed to an abnormal function of the anterior lobe of the pituitary which secretes the growth hormone. This hormone has an antagonistic effect on insulin by stimulating the alpha cells of the pancreas and by causing oversecretion of glucagon. The surgical removal of the anterior lobe of the pituitary or its destruction by other means could eventually alleviate hyperglycemia, acromegaly and the sequelae of hyperglycemia and may be considered the future treatment of choice. (20 references)

Alice R. Deutsch.

Boulin, R., Dollfus, M. A., Lebouch-Virgier, L. and Lebanc, R. **Sequelae of thrombosis of the cavernous sinus in two cases of diabetes.** Bull. Soc. belge d'opht. **115**:228-234, 1957.

The concomitant use of antibiotics and anticoagulants has had a favorable influence on the course of thrombophlebitis of the cavernous sinus, at least in most cases. Nevertheless the prognosis of this disease remains serious especially if it develops in diabetic individuals.

Two case histories are reviewed. Both patients were women, about 50 years old,

with diabetes of long standing, insufficiently treated. A minimal nasopharyngeal infection led to a thrombophlebitis of the cavernous sinus in spite of adequate antibiotic and anticoagulant treatment. In the first case a monocular thrombosis of the ophthalmic artery caused embolism of the homolateral central retinal artery and an insidious osteitis of the nose, still active after many months. In the second case hematuria necessitated interruption of the heparin treatment in spite of the presence of a thrombosis of the internal carotid with hemiplegia. Local complications were panophthalmitis and necrosis of the nasal orbital wall with sequestration. There was also a steady rise in insulin requirements.

Among 20 cases of thrombophlebitis treated during the last ten years two deaths occurred. One of these patients was a diabetic. The cause of death was a temporal lobe abscess which developed in spite of antibiotics and anticoagulants. The prognosis of cavernous sinus thrombosis (phlebitis) always should be guarded. (5 references) Alice R. Deutsch.

Dollfus, A. and Haye, C. **A survey of 360 juvenile diabetics (under 30 years of age).** Bull. Soc. belge d'opht. **115**:184-189, 1957.

The case histories of all diabetics examined at the eye department of the Hospitâl de la Pitié during the last ten years have been reviewed. Among more than 2,500 patients 360 were found to be under 30 years old and to have known about their disease for about 10 years. Seventy-eight of these patients showed abnormal eye conditions (21.6 percent). A distinct proportionate increase relative to the duration of the diabetes was found. Among the diabetics of 10 years' standing ocular abnormalities were found in 50 percent. After 18 years of diabetes eye diseases were seen in 90 percent of the patients. Alice R. Deutsch.

Evans, A. D., Pallis, C. A. and Spillane, J. D. **Involvement of the nervous system in Behçet's syndrome.** Lancet **273**:349-353, Aug. 24, 1957.

The prognosis in Behçet's syndrome is relatively benign except for the loss of vision that results from the recurrent keratoconjunctivitis. When the central nervous system is involved, the mortality is close to 50 percent and there is a discrepancy between the serious outcome of the disease and the demonstrable neuropathological findings. Attempts to transmit the virus to mice, guinea pigs and rabbits by the intracerebral, intramuscular, and intraperitoneal routes have failed. (3 figures, 20 references.

Irwin E. Gaynon.

Favory, A. and Lagroulet, J. **Optic atrophy and diseases of the retina in ocular onchocercosis.** Bull. et mém. Soc. franç. d'opht. **69**:392-407, May, 1956.

Onchocercosis presents a real health problem in extended regions of Africa and America. Nevertheless histopathologic studies have been made only rarely. The present study not only gives a detailed clinical description of a severe case of ocular onchocercosis but also constitutes the most complete pertinent histopathologic collection. Severe vascular changes were found in the region of the limbus, the retina, the choroid and the optic nerve. Living microfilaria were never observed on biomicroscopic examinations but disintegration products of microfilaria were seen in diseased corneas. Released toxic substances with a special affinity for the vascular system of the eye were considered to be a possible cause of the severe degenerative ocular lesions. The chorioretinal changes resemble those of trypanosomiasis; the disease of the optic nerve is similar to optic nerve lesions in arsenic poisoning and sleeping sickness. It seems interesting that living microfilaria were

found in tissues without any inflammatory or degenerative reactions. (9 figures)

Alice R. Deutsch.

Fischer, Franz. **The retinopathy of the young diabetic (under 40 years of age).** Arch. f. Ophth. **159**:246-256, 1957.

After a historical summary the author reports his findings in 61 diabetic patients between the ages of 14 and 39 years. There were very few less than 19 years old but then the incidence rises quickly. At all ages the clinical picture is dominated by three characteristics: 1. duration of the diabetes longer than 10 years, 2. arterial hypertension, and 3. a nephropathy, particularly the Kimmelstiel-Wilson lesion. The retinopathy is characterized by the same variations as that of the older diabetic. The hypertension and nephropathy as well as the retinopathy are all an expression of the specifically diabetic angiopathy. (90 references)

F. H. Haessler.

Mackie, E. G. **Paget's disease and the eye.** Tr. Ophth. Soc. U. Kingdom **76**:267-282, 1956.

The author emphasizes the relation of Paget's disease to loss of visual function in the aged. In many aged men it can be shown that osteoclastic osteoblastic elements are at work to distort bony structure. Paget's disease is found in three percent of all autopsies of subjects over the age of 40 years; the greatest incidence is in the lumbosacral and pelvic regions, and in the vertebrae and head in 50 and 28 percent respectively. The softening, distortion and remineralization with permanency of abnormal shape which afflicts long bones affects the head also with consequent osteoporosis or mushrooming of the skull and invagination of its base. Exudative macular disease, angioid streaks and senile macular degeneration are strikingly related to osteitis deformans.

The vascular character of the ocular complications in the aged is well known, as is the elevation of phosphatase in Paget's disease. Reis pointed out in 1915 that phosphatase in tissues where infiltration occurs is two to four times as active in the choroid as in the kidney. He also showed that the enzyme is located in the vascular endothelium in the retina and in the whole uveal membrane. The author points out that elderly people with Paget's disease may escape specific retinal damage yet may experience the ordinary fundus deterioration of their age and degree of sclerosis. However, if the circulation is sufficiently impaired a disciform or Junius-Kuhnt lesion may develop. (6 figures, 1 table)

Beulah Cushman.

Newell, Frank W. **Nevoxanthoendothelioma with ocular involvement.** A.M.A. Arch. Ophth. **58**:321-327, Sept., 1957.

Nevoxanthoendothelioma is a comparatively uncommon benign skin disease of infants in which discrete bright-orange nodules appear in the skin. Two case reports are given of ten months and two months old infants in which histologic study of tissue from the eye showed histiocytosis. One infant had glaucoma and the other a spontaneous anterior chamber hemorrhage. (5 figures, 9 references)

G. S. Tyner.

Palich-Szanto, O. and Bikich, G. **The diabetic vascular diseases of the eye.** Ophthalmologica **133**:109-118, Feb., 1957.

This search for characteristic clinical features of diabetes with retinopathy again reveals the significance of the duration of the disease, of the degree of control and a possible sex factor, women being more frequently affected than men. (2 tables, 18 references)

Peter C. Kronfeld.

Pimentel, P. C. and Pereira Machade, R. **Incidence of ocular manifestations of**

tropical diseases in the State of Rio de Janeiro and Espirito Santo. Arq. brasil. de oftal. **19**:295-298, 1956.

In this article various tropical diseases with ocular complications are listed which have been observed in these two states of Brazil. James W. Brennan.

Rodenhaeuser, Joe Henry. **Pigmentation of the eye in alkaptonuria.** Klin. Monatsbl. f. Augenh. **113**:202-215, 1957.

Report of a case of ocular pigmentation in each eye of a 61-year-old man is reported. Biopsy was obtained from the conjunctiva and the pigment appeared yellow-brown in hematoxylin and eosine. The author believes that ascorbic acid normally prevents the precipitation of alkaptonic pigments into the tissues. In-vitro experiments corroborate this assumption. The pigment appears in the eye only in those places which contain little ascorbic acid (pinguecula, sclera, cornea). (5 figures, 33 references)

Frederick C. Blodi.

Rodger, F. C. **Acute ocular onchocerciasis and its treatment.** Brit. J. Ophth. **41**:544-557, Sept., 1957.

In Africa onchocerciasis is seen rather commonly in various manifestations but most frequently as a limbitis, a sclerosing scleritis, an anterior uveitis or as a posterior exudative uveitis. Basically the African eye differs from the European eye in the frequency and density of patches of pigment both within and without the eye and of the frequency of pingueculae. Limbitis is quite commonly seen and is the result of invasion of the conjunctival space by microfilariae. It is relatively mild but may progress to a sclerosing keratitis with a characteristic pannus formation resulting in complete vascularization of the cornea. The uveitis is typical of a granulomatous infection; copious exudation with the organisms fills the pupillary space. The posterior infection is longlasting and very destructive of the posterior uvea. Treatment consists of an initial intravenous injection of 10 million units of T.A.B. vaccine followed by 10 gm. of diethylcarbamazine orally. This is quite effective and particularly so in the acute stages. (9 figures, 1 table, 8 references)

Morris Kaplan.

Vellieux, M., Rit, J. and LeBreton Oliveau, G. **Reflections on African ocular onchocercosis.** Ann. d'ocul. **190**:576-586, Aug., 1957.

The authors report their observations on 540 cases of ocular onchocercosis seen at the Institute for Tropical Ophthalmology in French West Africa. In contrast to other filariases, *Onchocerca volvulus* seems to have a predilection for ocular tissues. It is felt that there are many "healthy" carriers of microfilaria but that the full clinical picture is seen only in natives with marked malnutrition and avitaminosis.

The clinical signs of this disease are reviewed. The authors emphasize that the secondary glaucoma that develops is very difficult to control and fistulizing operations have been notoriously unsuccessful. At present a two-fold attack is being made on this disease. Local waterways are being treated with lindane products to destroy the insect vectors and the patients are given Notezine (a piperazine derivative) which is filariocidal and moranyl (a ureide complex) which acts on the adult encysted forms. Spectacular though the results may be in some cases the authors emphasize that there are many resistant cases and the final word on therapy has yet to be written. David Shoch.

Zanen, J., Szuchs, S. and Pirart, J. **Chromatic and achromatic thresholds in diabetes.** Bull. Soc. belge d'opht. **115**:210-219, 1957.

The present study involves the accurate measurement of the color thresholds of the foveal area. The scotometer presented before the French Ophthalmological So-

ciety in 1955 was the instrument of choice. The absolute and chromatic thresholds corresponding to wavelengths of 423, 495, 515, 591, 643 and 698 millimicrons were investigated. The subjective response of the individual patient was more or less hesitant because of the difficulties in exact color recognition on the borderlines. Eleven patients with diabetes and one with bronze diabetes (hemochromatosis) were examined. The central color scotoma supposedly characteristic for diabetes was only found in the bronze diabetic, but there was rather a general elevation of the achromatic thresholds of central vision, serving as evidence of a certain deficit of color vision in the diabetic. However, the elevations were not as prounounced as in patients with congenital protanopsias. (6 figures) Alice R. Deutsch.

19

CONGENITAL DEFORMITIES, HEREDITY

Badtke, G. **The histologic picture of a microphthalmic eye in dyscranio-pygophalangia.** Klin. Monatsbl. f. Augenh. **131**: 313-325, 1957.

This stillborn male infant had numerous anomalies of the skull, the lumbar part of the vertebral column, the extremities, the testes, the colon and other structures. The eyes were small with retinal colobomas, rosettes, persistence of hyaloid vessels and pupillary membranes, aniridia and defects in Descemet's membrane. The author believes that the anomalies of the neuroectoderm are the primary lesion which induce secondary mesodermal defects, such as aniridia. (9 figures, 12 references)

Frederick C. Blodi.

Cassady, J. R. and Light, A. **Familial persistent pupillary membranes.** A.M.A. Arch. Ophth. **58**:438-448, Sept., 1957.

Stahli is quoted as saying that the incidence of persistent pupillary membrane is 30.6 percent. As an example of a hereditary tendency, 18 members of four generations of a family are described, showing pupillary membrane remnants as the primary abnormality in 11 subjects, congenital cataracts in four and enlarged corneas in three. The authors suggest an explanation of the relationship of both the cataracts and the large corneas to the abnormality of the iris. (21 figures, 13 references) G. S. Tyner.

Joseph, Ruby. **A pedigree of anophthalmos.** Brit. J. Ophth. **41**:541-543, Sept., 1957.

A nine-months-old infant was found to have bilateral anophthalmos but seemed to be otherwise normal. Two brothers who had died in infancy were similarly affected. No other similar picture in family tree could be found indicating this to be a recessive characteristic. A family tree of four generations is presented. (1 figure, 6 references) Morris Kaplan.

Joseph, Ruby. **Congenital total cataract—possibly recessive.** Brit. J. Ophth. **41**:444-445, July, 1957.

Two sisters, aged 13 and nine years, had total congenital cataract and it is possible that a brother who died at one year of age was similarly affected. No other members of the family for two generations were so affected so that it was assumed that the pattern of inheritance was recessive. (1 figure, 1 reference)

Morris Kaplan.

Shepkalova, V. **Congenital melanosis of the eye.** Vestnik oftal. **5**:29-37, Sept.-Oct., 1957.

In this detailed review of the literature on the origin of the clinical features of congenital melanosis of the eyeball, the importance of the differential diagnosis is stressed. Pigmentation of the sclera is observed in ochronosis, a severe disorder of the general protein metabolism in which there is an abnormal dark pigmentation of the cartilages, ligaments and fibrous tis-

sues, and a dark color of the urine. Some eyes have been enucleated in ochronosis because malignant melanoma was suspected.

Congenital melanosis of the eye is met with mostly in dark-skinned people; it is usually unilateral. The pathologic picture of the sclera shows an accumulation of chromatophores, the cytoplasm being filled with melanin in all layers of the sclera. A male patient is described who had congenital melanosis of the right eye and in whom a malignant melanoma developed at the age of 24 years. Another patient had a periscleral melanoma in an eye with congenital melanosis. Pathologic findings are described in detail. A primary melanoma in the outer layers of the sclera and episclera is very rare. Out of 360 patients with malignant melanomas only two occurred in an eye with congenital melanosis. (5 figures in color)

Olga Sitchevska.

Wagner, Gunter. **Eye findings in premature children.** Klin. Monatsbl. f. Augenh. **131**:326-334, 1957.

The author examined 104 prematurely born children (birth weight between 1020 and 2500 gm.) at an age of three to five years. Four children, all girls, had severe defects (microphthalmus, lens luxation, RLF, buphthalmus). Three children, all girls, had hyperopia of more than 5 diopters, and 21 children had myopia of more than 3 diopters. (6 tables, 4 references)

Frederick C. Blodi.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Magitot, A. **Fiftieth anniversary. The service of Dr. V. Morax at the Hospital Lariboisiere.** Ann. d'ocul. **190**:474-490, July, 1957.

The first true service of ophthalmology at a hospital in France was apparently organized by Dr. Morax fifty years ago. This is a lively account of the development both of the man and the service. Dr. Morax is known chiefly for his interest in external infections of the eye and it is interesting to read that originally patients were divided into two groups: those with white eyes and those with red eyes. The former were sent to the refraction clinic, the latter to the laboratory for infectious diseases of the eye. A formidable photograph of Dr. Morax is shown in the article as well as three sketches of the floor plan of the eye service. (4 figures)

David Shoch.

Talkovsky, S. **Ophthalmologists in U.S.S.R. for 40 years.** Vestnik oftal. **5**: 16-20, Sept.-Oct., 1957.

The number of ophthalmologists in the U.S.S.R. increased in this proportion: in 1913, there were 300 oculists, 2,000 beds and 10 medical schools. In 1955, there were 7,285 oculists, 21,268 beds and 68 medical institutes with eye hospitals. The problem of training ophthalmologists is still a serious one. The best form of preparing qualified ophthalmologists is the three year residency in the institutes which trains them for later independent work. However, this form of preparation produces too few eye specialists. Therefore, shorter courses of a few months duration dedicated to the actual clinical problems of ophthalmology are practiced in many medical institutes. The better qualified oculists are sent to the periphery and less qualified ones are trained instead in the larger centers. The medical students are encouraged to select their specialty in ophthalmology while still in school by developing in them an interest in ophthalmology through discussion clubs of ophthalmologic problems. The need for increasing the number of ophthalmologists to cover the remote regions of the U.S.S.R. is still great.

Olga Sitchevska.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

DEATHS

Prof. Giuseppi Ovio of Rome, Italy, died in December, 1957, aged 95 years. His monumental history of ophthalmology, published in 1953 (two volumes, 2,336 pages), is the most definitive since that of Julius Hirschberg.

Dr. Carl Frederick Breisacher, Charleston, West Virginia, died October 16, 1957, aged 46 years.

Dr. Leon Clinton Combacker, Fergus Falls, Minnesota, died October 1, 1957, aged 74 years.

ANNOUNCEMENTS

OPHTHALMOLOGY JUBILEE PROGRAM

A special one-day scientific program will be presented by the Department of Ophthalmology of the Saint Louis University School of Medicine Thursday, March 27th, in honor of the 25th anniversary of its main teaching hospital, Firmin Desloge Hospital, Saint Louis. Ophthalmologists throughout the country are especially invited to attend the program which will be held in Miller Hall of the hospital. There will be no charges.

Guest speakers will include: Dr. Bernard Becker, professor of ophthalmology, Washington University, "The role of electrotonography in the management of glaucoma"; Dr. V. Everett Kinsey, assistant director of research, Kresge Eye Institute, Detroit, "Lens metabolism in normal and diabetic animals"; Dr. Paul A. Chandler, associate professor of clinical ophthalmology, Harvard University, "Practical considerations in the management of narrow-angle glaucoma." Dr. Elmer J. Ballintine, senior instructor in ophthalmology, Department of Surgery, Western Reserve University will be the guest speaker at the evening dinner meeting of the Saint Louis Ophthalmological Society which will conclude the session. A fifth guest speaker is to be announced.

Luncheon at Desloge Hospital will be provided free of charge. Send reservations to:

Dr. William H. Vogt, Jr., Chairman
Desloge Jubilee Committee
1325 South Grand
Saint Louis 4, Missouri

ORTHOPTICS COURSE

The basic course in orthoptics for technicians, sponsored by the American Orthoptic Council, will be held in the Department of Ophthalmology, University Hospital, University of Michigan, from June 23rd through August 16th. As usual, there will be didactic lectures and practical demonstrations, given by an outstanding faculty.

Additional information as well as application blanks may be obtained by writing:

Dr. John W. Henderson
Room 6155
Out-Patient Building
University Hospital
Ann Arbor, Michigan

COURSES IN BASIC OPHTHALMOLOGY

A comprehensive course in basic ophthalmology is offered by Wayne State University Graduate College of Medicine in co-operation with the Kresge Eye Institute. Set up in its present form nine years ago under the direction of Dr. A. D. Ruedemann, the course has constantly been improved and expanded. A new auditorium and a teaching laboratory at the Kresge Eye Institute have greatly improved facilities for didactic training. The roomy, well-equipped eye clinic in the Farwell Annex of Receiving Hospital makes possible thorough clinical training.

Prerequisites for the course are an M.D. degree from an accredited medical college and one year of rotating internship. Classes are held full-time for nine months; six mornings a week are taken up by lectures, five afternoons by clinics. Subjects covered are anatomy, bacteriology, embryology, histology, pathology, ophthalmic biochemistry, ophthalmoscopy, physiologic optics, refraction, and ocular motility. Other related subjects are also briefly surveyed: such as, nutrition, endocrinology, and so forth. The principles of surgery are taught by lectures and laboratory work on animals' eyes. Tuition is $300.00.

Five fellowships at Detroit Receiving Hospital are available each year. Harper and Wayne County General Hospitals send their residents in ophthalmology to Wayne State University Graduate College of Medicine for this training during their first year of residency. Tuition for fellows and residents in affiliated hospitals is only $100.00.

Openings are available for the academic year 1958-59. Classes begin on September 8, 1958.

Further information may be obtained from:

Dr. A. D. Ruedemann
690 Mullett Street
Detroit 26, Michigan

TRAVEL FUND AVAILABLE

The National Institute of Neurological Diseases and Blindness announces the establishment of a special travel fund designed to aid younger ophthalmologists and other scientists in ophthalmic teaching or research to attend the XVIII International Oph-

thalmology Congress in Brussels, September 8th to 12th. The fund was made possible by a grant from the National Advisory Neurological Diseases and Blindness Council to a council subcommittee. In authorizing the grant, the council noted that "an international congress offers enormous value in the training and exchange of ideas" among scientific investigators.

The subcommittee asks that all applications for aid from the fund be made by letter and addressed to Dr. Gordon H. Seger, Chief, Extramural Programs, National Institute of Neurological Diseases and Blindness, Bethesda, Maryland. The letter of application should also provide a brief review of the applicant's educational and occupational background and should indicate his major field of interest. The deadline for receipt of applications is March 1, 1958. The council subcommittee will make its selections and notify those to be aided by letter no later than April 1, 1958.

Orthoptic course offered

The Kresge Eye Institute again offers its facilities for the training of orthoptic technicians. This program is open to any young woman over 20 years of age, who has successfully completed at least two years of college or is a graduate nurse. Good health and vision are required of the trainees.

The course consists of 12 months' didactic and practical training. Didactic training, which is part of the basic ophthalmology course for doctors at Wayne University Graduate College of Medicine, will be given five mornings a week for the first semester. Lectures on refraction, retinoscopy, physiologic optics, perimetry, ocular anatomy, neuroanatomy, and motility are given by members of the Kresge Eye Institute staff and professors of Wayne State University College of Medicine.

Practical training must begin in mid-August, one month prior to the beginning of the lecture courses. This practical training continues for one year, at which time students are eligible to take the board examinations for certification given by the American Orthoptic Council orally in August and in writing each fall.

Limited financial assistance is available for some students who require it. Requests may be made either through Kresge Eye Institute or the Delta Gamma Fraternity (Mrs. H. L. Marchant, 4006 Virgilia, Chevy Chase 15, Maryland).

For further information write to:
Dr. A. D. Rudemann
Kresge Eye Institute
690 Mullett Street
Detroit 26, Michigan

Oregon academy

The 17th annual spring convention of the Oregon Academy of Ophthalmology and Otolaryngology will be held in Portland, Oregon, March 21st through 25th. The otolaryngology sessions will be held on March 21st and 22nd. For the ophthalmology sessions, March 24th and 25th, the speakers will be: Dr. W. Morton Grant, Boston; Dr. S. Rodman Irvine, Los Angeles; and members of the Department of Ophthalmology, University of Oregon Medical School, headed by Dr. Kenneth C. Swan. For further information write:

Dr. Robert W. Zeller
603 Mayer Building
Portland 5, Oregon

Gill congress

The 31st annual spring congress in ophthalmology, otology, rhinology, laryngoscopy, faciomaxillary surgery, bronchoscopy, and esophagoscopy will be held at the Gill Memorial Eye, Ear, and Throat Hospital and the Elbyrne G. Gill Eye and Ear Foundation, Roanoke, Virginia, April 14th through 19th.

Among the guest speakers will be Dr. David B. Allman, Atlantic City, president of the American Medical Association. Guest speakers in ophthalmology will be: Dr. Roscoe J. Kennedy, Cleveland; Dr. Donald J. Lyle, Cincinnati; Dr. Edward W. D. Norton, New York; Dr. Donald M. Shafer, New York; Dr. Byron Smith, New York; Dr. Richard C. Troutman, New York; Dr. Henry P. Wagener, Rochester, Minnesota; and Dr. Lorenz E. Zimmerman, Washington, D.C.

Miscellaneous

Minnesota continuation course

Guest speakers at the continuation course in ophthalmology for specialists, University of Minnesota, were Dr. Harold W. Brown, New York; Dr. Hermann M. Burian, Iowa City; and Dr. Harold G. Scheie, Philadelphia. The course was presented under the direction of Dr. Erling W. Hansen, head of the Department of Ophthalmology, and the faculty included members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

Cruise and scientific congress

More than 400 physicians and members of their families embarked on the *Queen of Bermuda* in New York on February 1st for a two weeks' combination cruise and scientific congress sponsored by the Pan-American Association of Ophthalmology. The program consisted of symposia, workshop sessions, motion pictures, and visits to medical institutions in port cities.

Dr. John M. McLean, New York, was the leader of a symposium on glaucoma; Dr. Walter H. Fink, Minneapolis, one on strabismus; Dr. James E. Lebensohn, Chicago, one on refraction problems; Dr. Conrad Berens, New York, one on the complications of cataract surgery; Dr. William L. Benedict, Rochester, Minnesota, one on lesions of the orbit.

Leaders of the workshop sessions were: Dr. Walter S. Atkinson, Watertown, New York, on ocular anesthesia; Dr. Elizabeth Constantine, New York, on orthoptics; Dr. Henry L. Birge, Hartford, Connecticut, and Dr. James H. Allen, New Orleans, on treatment of ocular infections.

Wills Eye Hospital

The 10th annual clinical conference of the staff and the Society of Ex-Residents of Wills Eye Hospital, Philadelphia, was held on February 20th, 21th, and 22nd. Wills Eye Hospital is presently celebrating the 125th anniversary of its founding.

Eye-Bank established

The Eye-Bank and Sight Conservation Society of Virginia has been established at the Elbyrne G. Gill Eye and Ear Foundation, 711 South Jefferson Street, Roanoke, Virginia, under the sponsorship of the Lions Club of Virginia.

Societies

Oxford congress

The 43rd annual meeting of the Oxford Ophthalmological Congress will be held at the lecture hall of the School of Philosophy, South Parks Road, Oxford, England, on July 7th, 8th, and 9th. Registration will be on the evening of Sunday, July 6th, at Balliol College, Broad Street. On Monday, there will be a discussion on "The influence of vascular changes in progressive failure of vision." On Tuesday morning, the Doyne Memorial Lecture will be delivered by Mr. O. Gayer Morgan of London. The subject of the lecture will be "The early clinical diagnosis of glaucoma." On Wednesday morning, the discussion will be "The etiology and treatment of uveitis."

Ophthalmologists not already members of the congress who wish to attend the meetings should write to:

Ian C. Fraser
Honorary Secretary
21 Dogpole
Shrewsbury

Congress of allergy

The International Association of Allergology and The French Allergy Association will sponsor the III International Congress of Allergy to be held in Paris, France, October 19th through 26th. For further information regarding the program and the papers to be presented write to:

Dr. Samuel M. Feinberg
303 East Chicago Avenue
Chicago 11, Illinois

Kansas City society

Guest speaker at the January meeting of the Kansas City Society of Ophthalmology and Otolaryngology was Dr. Harold F. Falls, Ann Arbor, Michigan. Dr. Falls presented the Curran Lecture. His subject was "Developmental anomalies of the ocular segment and their significance and management." Dr. Falls also discussed "Unusual ophthalmoscopic changes in diagnostic significance." Other speakers at the ophthalmology session were Dr. John Bradford, Dr. Dick Underwood, and Dr. Larry Calkins.

Madrid society

Newly elected officers of the Ophthalmological Society of Madrid are Dr. Marin Amat, president, Dr. Tena Ibarra, treasurer, Dr. Arjona, vice-president, Dr. del Rio Cabanas, secretary, and Dr. Leoz de la Fuente, director.

Brooklyn meeting

At the 146th regular meeting of the Brooklyn Ophthalmological Society, Dr. John F. Gipner presented a paper on "Modern trends in ophthalmic surgery," which was discussed by Dr. Richard C. Troutman. Dr. Girolamo Bonaccolto presented a motion picture on "Dacryocystorhinostomy." Officers of the society are: Dr. Rodolph M. Cutino, president, Dr. Ira W. Mensher, vice-president, Dr. Nicholas P. Tantillo, secretary-treasurer, and Dr. A. Benedict Rizzuti, associate secretary-treasurer.

Ophthalmologic allergy

The newly elected officers of the American Society of Ophthalmologic and Otolaryngologic Allergy are: President, Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19, Pennsylvania; seccretary-treasurer, Dr. Daniel S. Destio, 121 South Highland Avenue, Pittsburgh 6, Pennsylvania. The next meeting of the society will be at the Palmer House, Chicago, on October 16th and 17th.

College of Surgeons meetings

New York

The New York sectional meeting of the American College of Surgeons will be held in New York on March 3rd through 6th. The program for ophthalmology includes a symposium on Monday, March 3rd, from 9:00 A.M. to 12:00 noon, at the Waldorf-Astoria Hotel, on "Extraocular muscles." The subjects to be discussed and the participants are: A. Gerard DeVoe, New York, presiding; "New concepts of extraocular muscle physiology," Goodwin M. Breinin, New York; "Diagnosis and treatment of convergent squint," Hermann M. Burian, Iowa City; "Exotropia: Diagnosis and treatment," Philip Knapp, New York; "Diagnosis and treatment of vertical muscle deviation," Marshall M. Parks, Washington, D.C.

On Tuesday, March 4th, there will be a symposium on "Retinal detachment," from 9:00 A.M. to 12:00 noon at the Waldorf-Astoria, with John H. Dunnington, New York, presiding. Joseph A. C. Wadsworth, New York, will discuss "The physiopathology of retinal detachment"; Graham Clark, New York, "Selection of operative procedures for retinal detachment; Charles L. Schepens and Ichiro D. Okamura, Boston, "The scleral buckling procedures: Results"; Donald A. Shafer, New York, "Implantation of vitreous."

Salt Lake City

The Salt Lake City sectional meeting will be held on March 17th through 19th. The following program for ophthalmic surgeons has been arranged:

"Keratoplasty in treatment of endothelial dystrophy," Max Fine, San Francisco; "Factors modifying prognosis in keratoplasty," Leonard Christensen, Portland; "Glaucoma: Medical control measures and where effective," Robert N. Shaffer, San Francisco; "Control of too-soft eye following glaucoma surgery," Orwyn H. Ellis, Los Angeles; "Pathologic reasons for failure in glaucoma surgery," A. Ray Irvine, Jr., Los Angeles.

A. Ray Irvine will be the moderator of a panel discussion on "Choice of surgical procedure in glaucoma." The collaborators will be Leonard Christensen, Max Fine, Orwyn Ellis, and Frank C. Winter, Menlo Park. On the panel for the symposium on "Choice of procedure in retinal detachment," will be Frank C. Winter, Carroll Weeks, Los Angeles, and Daniel G. Vaughn, San Jose.

New York conference

The annual participating conference of the New York Society for Clinical Ophthalmology will be held on March 28th and 29th at the Waldorf-Astoria Hotel. Registrations are still being accepted by Miss Gloria Benabe, 737 Park Avenue, New York. The fee of $50.00 includes two luncheons.

New York alumni

The alumni and staff of The New York Eye and Ear Infirmary, 218 Second Avenue, New York, will hold their annual spring meeting from April 21st through April 24th. In the mornings seminars will be held on "Ocular pathology," "Retinal detachment surgery," "Glaucoma," "Ocular injuries," "Cataract surgery," and "Motility." Clinical courses and closed-circuit television programs will be given in the afternoons.

Seminar on glaucoma

A seminar on glaucoma with particular emphasis on gonioscopy and the study of the anterior angle will be given at the Brooklyn Eye and Ear Hospital on May 19th, 20th, and 21st. Ample opportunity for practical instruction in the use of the gonioprism will be given and material from the glaucoma clinic will be utilized.

The course will be given by Dr. Daniel Kravitz, assisted by Dr. Mortimer A. Lasky, Dr. A. Benedict Rizzuti, Dr. Abner S. Rosenberg, Dr. Nicholas P. Tantillo and Dr. Samuel Zane.

Registration is limited to six (6) ophthalmologists only.

Application and the fee of $50.00 may be addressed to:

Dr. Daniel Kravitz
Brooklyn Eye and Ear Hosiptal
29 Greene Avenue
Brooklyn 38, New York

Prevention of Blindness meeting

The National Society for the Prevention of Blindness will hold its annual Sight-Saving Conference at the Bellevue-Stratford Hotel, Philadelphia, March 12th through 14th.

Personals

Dr. J. Reimer Wolter, formerly associated with the University of Michigan, Ann Arbor, has been appointed an "Oberarzt" at the Eye Clinic of the University Hospital in Frankfurt am Main, West Germany. Dr. Wolter assumed his new duties on February 1st.

NEW ORLEANS ACADEMY OF OPHTHALMOLOGY

The Eighth Annual meeting of the New Orleans Academy of Ophthalmology will be held in New Orleans in the Roosevelt Hotel—February 24-28, 1958, featuring "Symposium on Uveitis." The registration fee of $75.00 includes associate membership in the Academy for the year 1958, as well as all other features of the convention. Hotel reservations should be made early by writing directly to the Executive Secretary, P.O. Box 469, New Orleans, La.

Urgent Request

The Uveitis Laboratory, University of California School of Medicine, San Francisco, is interested to obtain freshly enucleated eyes from patients with *all types of uveitis* and other endogenous inflammations. Attempts are being made to isolate etiologic agents from these eyes.

The eyes should *not* be fixed in preservatives or frozen, but placed in a sterile bottle, packaged, and shipped as quickly as possible. Please send specimens air express, special delivery, *collect*. Enclose history and findings and mark the package "Fresh Tissue Specimen—Rush."

A report of isolations of organisms and pathologic findings, including a slide, will be sent to the contributor. Credit will be given in any resulting publications if desired.

• • • •

Telegraph collect if specimen being sent. Send eyes to Samuel Kimura, M.D., Michael J. Hogan, M.D., or Phillips Thygeson, M.D., University of California School of Medicine, San Francisco 22.

ANNALES D'OCULISTIQUE

One of the oldest ophthalmological reviews. Founded in 1838 by Florent Cunier, continued by Warlomont (1853), Valude and Sulzer (1891), V. Morax (1898).

The publication includes original articles, notes on practical ophthalmology, descriptions of clinical cases and reports of the proceedings of European ophthalmological Societies.

This review is published by Messrs. Doin & Co.
8 Place de l'Odeon Paris 6e

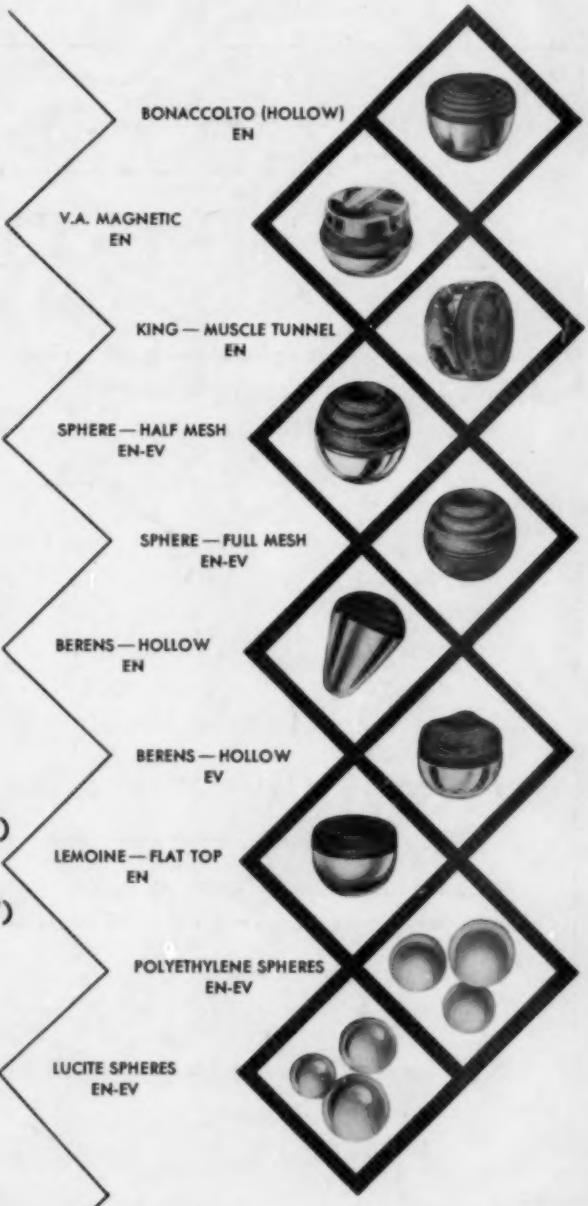
Subscription rates: France and Colonies—4000 Frc.
Abroad —4200 Frc.

AMERICAN
OPTICAL
COMPANY

MONOPLEX

IMPLANTS

for
ENUCLEATION (EN)
and
EVISCERATION (EV)



For information and operative procedures, write to
MONOPLEX MANAGER
AMERICAN OPTICAL COMPANY
Southbridge, Mass.

American Optical
COMPANY